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THE INFLUENCE OF POSTURE ON DIGESTION IN INFANCY *

CHARLES HENDEE SMITH, M.D.
AND
LEON THEODORE LEWALD, M.D.
NEW YORK

Success in infant-feeding depends not only on the composition of the food, but also on the way in which it is given. The manner of feeding and the management of the child between feedings have received scant attention, yet there are certain points of considerable importance in this connection disregard of which may result in unsuccessful feeding, no matter how carefully the food is prescribed.

Posture has an important bearing on digestion in infancy. For many years the teaching of doctors and nurses has been that an infant must be put to sleep in the horizontal position immediately after feeding. Many mothers, however, hold the child upright after feeding before putting him down to sleep. It is believed that one of these plans is correct and the other wrong, both in theory and practice, and that the Roentgen ray has demonstrated the truth of this. The way in which posture can affect digestion depends on two main factors.

The first factor concerns the anatomy of the stomach. Roentgenography has shown the fallacy of the old idea that the stomach in infancy is vertical in position. There are several "types" of stomach which resemble the adult types in a general way, but the shape of the stomach depends on the amount of food and gas present, pressure from the outside, posture, etc. These different types have no especial importance. There are certain anatomic features which have to be borne in mind. The esophagus enters the abdomen by an opening in the diaphragm, almost in the mid-line, in contact with the aorta and the ventral surface of the bodies of the vertebrae. The cardiac orifice lies just to the left of this position and the cardia is held firmly to the posterior abdominal wall by the esophagus as it pierces the diaphragm. The fundus of the stomach lies in the left paravertebral groove, that is,

* Received for publication Jan. 25, 1915.

* Presented in part at the Pediatric Section, N. Y. Academy of Medicine, April, 1914. From the Pediatric Division and Roentgen-Ray Laboratory, St. Luke's Hospital.

in the lateral recess formed by the projection forward of the vertebral column into the abdominal cavity. The fundus reaches a plane considerably dorsal to the cardiac orifice. It is covered in front only by the liver and extends to the anterior abdominal wall. The pyloric end of the stomach is pushed forward by the vertebral column, pancreas and duodenum. The pyloric opening is directed backward toward the duodenum, but lies in a plane which is ventral to that of the cardia. The cardiac opening is behind the center of the anteroposterior diameter of the stomach.

The second factor is that there is a certain amount of gas in the stomach of every individual. This is a matter of common knowledge, but has been emphasized by the Roentgen ray. Practically every roentgenogram taken in the erect position shows a bubble of gas in the fundus end of the stomach (*Magenblase*). In the prone position it is not possible to estimate accurately the amount of gas present, as will be shown later. This gas may be derived from three sources, namely, swallowed air, gastric fermentation and intestinal gas.

Gastric fermentation is rare and is an important source only when there is stagnation from obstructed pylorus or from gastric atony of extreme degree. Intestinal gas does not commonly pass back into the stomach in all probability. Swallowed air accounts for the gas in the stomach in the large majority of cases. Air-swallowing takes place constantly, though to a variable degree. The size of the pharynx is such that it is rarely filled completely with food at the time of each swallowing act, and its anatomic relations prevent its collapse on a small amount of fluid. Air is constantly present in the pharynx and some air is forced down with the food by the contraction of the swallowing muscles. It is often possible to hear a child gulp down air with each swallow of milk. More or less air may be swallowed with saliva or nasal or bronchial mucus between feedings. Tongue-suckers, thumb-suckers and babies with a "comforter" habit suck and swallow air a considerable part of their waking hours. Infants fed on liquids swallow more air than older children fed on solids or semisolids. The more solid food is swallowed in a bolus and more completely fills the pharynx, while liquids are swallowed a few drops at a time. The smaller amount of air swallowed with semisolids may possibly be the explanation of the cessation of vomiting in the refractory cases in which no liquids are retained, but semisolid porridge is retained.

Usener,[1] in a paper on *Aerophagie*, noted the air-bubble in older children to be smaller than in infants. He stated also that the gas in the stomach has been proved to contain only oxygen, nitrogen and carbon dioxid or merely the constituents of air. He explained the mechanism

1. Usener: Ztschr. f. Kinderh., 1912, v. 440.

of the comparatively large amount of air swallowed by infants as follows: The horizontal position is probably one cause of air-swallowing, as it is in adults in bed with acute illness or after operations who often have great difficulty in swallowing properly and often swallow air with liquids. In nurslings each small drop of milk on reaching the region where the swallowing reflex is initiated starts the reflex and causes swallowing of air with the milk.

Flesch and Peteri,[2] with the fluoroscope, saw an air-bubble appear with the first swallow and fill one-third to one-fourth of the stomach during the feeding in both breast-fed and bottle-fed infants.

Grulee[3] thinks that the gas mostly comes from gastric fermentation due to stagnation from short-interval feeding. If this were true, the air-bubble should be absent when children are fed at long intervals. In our observations such children' however, show gas-bubbles but little smaller than those fed too often. The character of the eructated gas suggests fermentation only in cases of extreme gastric stagnation.

Figures 1, 2 and 3 demonstrate the source of the gas in the stomach. This infant was given her usual bottle with bismuth 1:8. She took the first half ounce in about two minutes. Three minutes later the first exposure was made (Fig. 1). It shows a lateral view of the child held in the horizontal position. There is a small amount of milk in the stomach with a moderate bubble of gas between it and the anterior abdominal wall. The child was held in the horizontal position and then given 1½ ounces more of the food in three minutes. Figure 2, taken at ten minutes from the beginning of the feeding, shows a very large amount of gas above (ventral to) the milk. The stomach is greatly distended already. The child was crying and was uncomfortable even with half of the feeding in the stomach. The child was then given 1½ ounces more, but this was taken badly and the last half ounce of the feeding was absolutely refused. About fifteen minutes were spent in trying to get the food down; different nipples were tried, but the child could swallow no more. Figure 3 — eighteen minutes after Figure 2 — shows why the last half ounce was refused. The enormous gas-bubble plus the milk had so distended the stomach that no more could be taken. This gas must have been swallowed air, since it has appeared during the feeding, there was no stagnation of food from the last meal, and the gas appeared too rapidly to be the result of fermentation. The food contained no substance which could form gas with the gastric juice in amount large enough to distend the stomach to this extent. The bismuth subcarbonate used does not evolve gas

2. Flesch and Peteri: Ztschr. f. Kinderh., 1911, p. 227.
3. Grulee: Infant Feeding, Saunders, 1914.

Figure 1

Figure 2

Figure 3

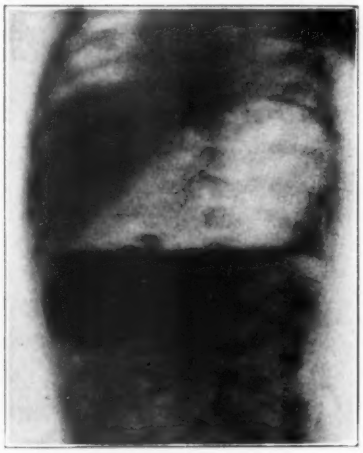

Figure 4

Figure 5

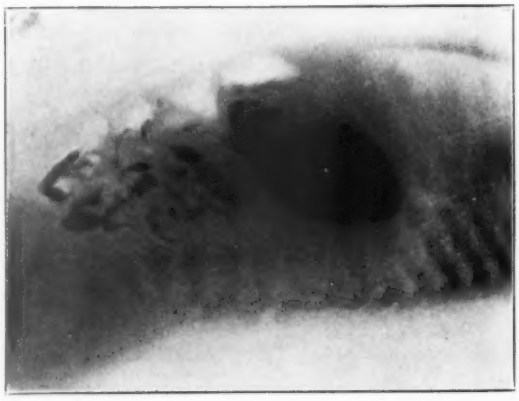

Figure 6

Figure 1.—Child, aged 4 months; shown also in Figures 2, 3, 4, 5 and 6. History of vomiting since birth, on breast and bottle. Nursed eight weeks. Given usual feeding, that is, fat 2 per cent., lactose 6 per cent., protein 1.75 per cent. Four ounces every three hours, six feedings a day. To this ½ ounce of bismuth subcarbonate was added. One-half ounce was taken in the first two minutes. Plate taken five minutes after feeding was begun. Supine position, left side against the plate. There is a small amount of milk in the stomach, with a small bubble of gas anterior to it. There is much gas in the small intestine.

Fig. 2.—The child was then given 1½ ounces more food in three minutes. Plate taken ten minutes after feeding was begun. Supine position, left side against the plate. There is a large air bubble in front of the milk, which has gravitated to the back part of the stomach. A little bismuth has entered the small intestine. Note the position of the esophagus.

Fig. 3.—The child was then given 1½ ounces more of the feeding. This was taken with difficulty, and the remaining ½ ounce was refused. Child seemed in pain, cried and regurgitated a small amount. Plate made twenty-eight minutes after feeding was begun. Position same as above. Enormous bubble of air distending stomach. Note double pouch of fundus and pyloric end of stomach (as in cases of dilatation) in both Figures 2 and 3.

Fig. 4.—Thirty minutes after feeding was begun. Erect position, abdomen against the plate. Milk has gravitated to the lower part of the stomach, forcing air to the upper part. After this plate there was a large eructation of gas. The child at once stopped crying and went to sleep.

Fig. 5.—Thirty-five minutes after feeding was begun. Position same as in Figure 4. Air bubble has disappeared. Note migration of pylorus toward left, as compared with Figure 4.

Fig. 6.—Thirty-seven minutes after feeding was begun. Supine position, left side against plate. The stomach has contracted on the food; little or no gas remains. There is one inch difference in the anteroposterior diameter of the abdomen in Figures 3 and 6. A good deal of bismuth has entered the small intestine. The double pouch appearance seen in Figure 3 is gone.

with hydrochloric acid under 0.5 per cent. This gas-bubble must be composed largely if not entirely of swallowed air.

Food taken by a child enters the stomach and gravitates to its lowest part, displacing the gas present to the highest part. The behavior of liquid and gas in the stomach does not differ from that of liquid and gas in a glass bottle or any other receptacle. When the child is held in a horizontal position the liquid food therefore gravitates to the posterior part of the stomach, that is, the fundus end, and forces the gas present to the anterior part of the fundus and to the right end of the stomach. Since the cardiac orifice is situated well back against the vertebral column, it follows that it will be covered by the liquid and that the gas cannot escape into the esophagus. It can escape only through the pylorus, if at all. If the amount of food plus gas is enough to distend the stomach, either food and gas must be forced out through the pylorus, or regurgitation of food must take place through the esophagus until the tension on the stomach wall is relieved. The horizontal position causes a perfect water-lock to form as far as the gas and the cardiac opening are concerned. This may be readily imitated by taking a bottle two-thirds full of liquid. When the bottle is held on its side no air can escape through the bottle mouth. When the bottle is held upright, the gas can escape with ease if under pressure.

When a child is held upright the liquid gravitates at once to the dependent part of the stomach displacing the gas to the highest part — that is, to the upper part of the fundus — and this makes it possible for the gas to escape by the esophagus. This often takes place at once as soon as the child is held erect.

Figures 3 to 6 show the effect of posture. Figure 3, as stated above, shows the enormous distention of the stomach with gas in the horizontal position. Two minutes later the child was held upright and Figure 4 shows the milk at the lower, the gas at the upper part of the stomach. The child was then held against the mother's shoulder, patted gently on the back and at once eructated a large amount of gas. He then became perfectly quiet, stopped crying and dozed. Figure 5 — five minutes later — shows that the gas has been almost entirely eructated and that the stomach has contracted on the food. Figure 6 — two minutes later — shows again a lateral view, in the supine position. There is only a small bubble of gas against the anterior abdominal wall. The rest of the stomach has contracted on the food. The pyloric end shows well and there is a good deal of bismuth in the intestines. The comparison between Figures 3 and 6 is most instructive, and corresponded to the physical signs in the epigastrium and to the child's comfort. To one seeing only the picture shown in Figure 6 it would not seem possible that the same stomach could hold the amount of gas shown in Figure 3.

Figures 7, 8, 9 and 10 also demonstrate what happens when an infant is fed in the ordinary horizontal position and is allowed to remain in that position. This child was a normal healthy child of 6 months in the hospital while his mother was undergoing an operation. He was given his usual bottle with one-half an ounce of bismuth subcarbonate, which he took readily and easily. During the feeding he was kept in the horizontal position. His abdomen, which was moderately distended before feeding, became much more so during the feeding and the child showed every sign of acute discomfort. He began to cry, squirmed as if in pain and at the time of the first exposure, three minutes after feeding, began to regurgitate milk. This took place easily without any violent vomiting effort.

The Roentgen ray shows the reason for these phenomena. Figure 7 shows a lateral view of the abdomen in the supine position. The milk in the stomach has gravitated into the posterior part and has displaced the gas to the anterior part of the stomach. The esophagus shows plainly as the child was regurgitating during the exposure. Its position well below the level of the milk shows plainly. It is perfectly evident that no gas can possibly escape from the esophagus until enough milk has been vomited to uncover the cardiac orifice of the stomach.

The child was then held erect and the second exposure two minutes later shows the immediate effect of gravity. The milk has gravitated to the bottom of the stomach, forcing the gas to the upper part of the fundus, and it was now *possible* for the first time for the gas to escape. After this exposure the child was patted on the back rather forcibly and he eructated gas several times. This was done easily and no milk was vomited. The entire aspect of child changed at once. He stopped crying and went to sleep. Figure 9 shows his stomach five minutes later (ten minutes after feeding). The stomach has lost all of its gas except a small bubble just at the cardia, which is overlapped by the liver shadow. The stomach has contracted down on the milk and small amounts of bismuth have entered the small intestines. The child was then again put on its back. Figure 10 shows the stomach contracted down on the milk and illustrates its oblique position from above downward and forward to the right. The contrast between Figures 7 and 10 is as striking as was the contrast in the aspect of the infant.

The horizontal prone position is better than the supine, because the liquid food gravitates to the anterior part of the stomach and forces the gas to the posterior part. This places the cardiac orifice above the level of the fluid, and makes it possible for the gas to escape by the esophagus. This is perhaps the reason why so many infants sleep better "on their stomachs" than on their backs.

Figure 7

Figure 8

Figure 9

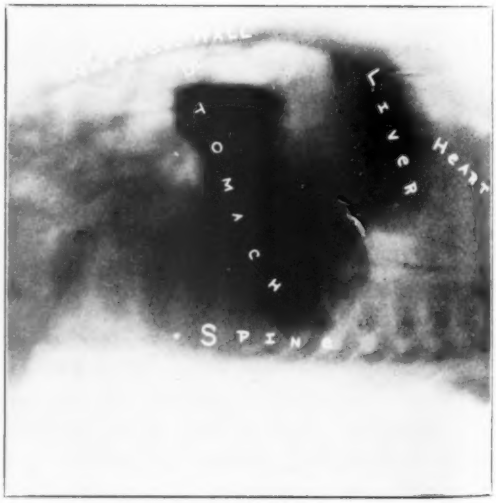

Figure 10

Fig. 7.—Healthy child, aged 6 months; shown also in Figures 8, 9 and 10. Given usual feeding of 6 ounces plus ½ ounce of bismuth subcarbonate. Feeding taken eagerly in ten minutes; considerable air swallowed with it. Position horizontal during and after feeding. Plate made three minutes after feeding. Child held in supine position, left side against the plate. Child very uncomfortable; vomited during first exposure. Milk and bismuth are in the posterior part of the stomach, gas in the anterior part. Esophagus is shown well below the level of the fluid, lying on the bodies of the vertebrae. Considerable gas in the intestine.

Fig. 8.—Five minutes after feeding. The child was held in the erect position, the anterior abdominal wall held against the plate. The gas has been forced upward to the upper pole of the stomach by the fluid which has gravitated to the lower part. After this plate the child was held upright, and was patted on the back. He eructated gas twice in considerable amounts. He immediately became more quiet, stopped crying and went to sleep.

Fig. 9.—Ten minutes after feeding. Position same as last plate. The gas has all been expelled from the stomach except a very small bubble just at the cardia, that is, just to the left of the vertebral column. The stomach is contracting down on the food. The splenic flexure contains a good deal of gas. This was overlapped by the stomach in the last picture.

Fig. 10.—Fifteen minutes after feeding. Position supine, left side against the plate, as in first plate. The stomach has contracted, and a peristaltic wave constricts it in the middle. A very small bubble of gas lies between the level anterior line of the fluid and the anterior abdominal wall.

This is shown in the third series of radiograms. Figure 11 shows the same large air-bubble in the anterior part of the stomach, the child being in the supine position. The child was turned over into the prone position and the illustration shows the air-bubble forced back by the milk which now occupies the anterior part of the stomach (Fig. 12). The position of the esophagus does not show, as the child was not vomiting, but it can be determined by noting the bodies of the vertebrae, and remembering that the esophagus lies about 1 cm. from their ventral surface. This is well above the level of the liquid.

The third picture (Fig. 13) shows the child held erect. He eructated some at once so that the bubble in the fundus end is smaller than in the last plate. He was then placed face downward and a plate made with the rays directed from behind forward (Fig. 14). This is the usual prone picture and shows how impossible it is to estimate the amount of gas present in this position, as it overrides the milk and only shows as a rim at the fundus end. It also shows the mobility of the pyloric end of the stomach, which is here pushed well over to the right of the mid-line (Contrast Fig. 13). The effect of posture and pressure has made two apparently different "types" of stomach.

The child was then held up and eructated gas twice, without vomiting. The next picture (Fig. 15) shows a decidedly smaller air-bubble. The last picture (Fig. 16) taken from right to left in the supine position shows the fundus end of the stomach contracted down on the milk and only a small bubble present toward the pyloric (anterior) part of the stomach. The contrast between Figures 11 and 16 is again striking enough to account for a good deal of difference in the comfort of the child.

If the child is kept horizontal from one feeding to the next the result depends on how much air is swallowed with the food, the capacity of the stomach and the ease with which the child vomits. If a large amount of air is swallowed, vomiting nearly always takes place, usually in the form of simple regurgitation, but occasionally the vomiting may be violent and projectile. Children vary greatly in the amount of air swallowed and in the degree to which the stomach will bear distention. Some will swallow and retain an enormous air-bubble without vomiting, but the usual result is rejection of part of the food by mouth until the tension on the stomach wall is relieved. The effect on the stomach of the extreme distention seen in some cases must be unfavorable to digestion. It seems as if there must be interference with secretion by the pressure on the mucosa and blood vessels. If the gas is expelled, then the stomach is free to contract and secrete. When the child is *kept* in the horizontal supine position the gas is gradually forced through the pylorus, and must traverse the intestine before escaping,

for even if part of the food is vomited the gas cannot escape by mouth. If the gas is in large amount it causes intestinal colic. This symptom may often be entirely relieved by assisting the expulsion of the gas by mouth, so that it need not be passed through the intestine. The symptom of "colic" is probably due in many cases to painful gastric distention, as well as painful intestinal peristalsis. The frequency with which colic accompanies regurgitation is suggestive of their common origin.

Figures 17 to 20 illustrate this point. Figure 17 shows the gas-bubble as usual at the anterior part of the stomach. The child eructated all the gas as soon as he was held upright, so Figure 18 shows no gas in the stomach. Figure 19, in the prone position, shows the change in shape and position of the stomach due to pressure on the abdomen, compared with Figure 18. Figures 17, 18 and 19 all show a large amount of gas in the small intestine. In Figure 20 — five hours and twenty-five minutes after feeding — there is still bismuth in the stomach. This child was fed every three hours, so that the second feeding was taken before the stomach was empty and mixed with the residue of the first. The child should have been fed every four hours. The cecum contains almost all of the rest of the bismuth except a small amount in the small intestine. This corresponds to the usual mechanism of a rapid passage of food through the small intestine. The transverse colon is full of gas, namely, that which was in the small intestine in Figures 17, 18 and 19. There is very little gas in the small intestine. This is in accordance with the fact that the gas taken in with the bismuth meal was expelled by mouth, and therefore was not passed through the intestine as would have been necessary if the child had been kept in the horizontal position.

Children vary greatly in their ability to relax the cardia, and eructate, some doing it easily and apparently voluntarily, while others do it only with great difficulty. The instinctive posture which many mothers assume (if not taught otherwise by nurse or doctor), when they hold the baby up against the shoulder after nursing, is the one in which the baby can most easily get rid of this undesirable swallowed air. Patting on the back aids the process of eructation, and it sometimes requires patting of considerable force. Slight pressure on the epigastrium may be needed, the mother grasping the baby's abdomen with her free hand and gently rubbing and pressing the stomach with her thumb, as he rests against her shoulder. At times the eructation may be violent and the contraction of the stomach so forcible and rapid that a small amount of milk comes up with the air. The amount of regurgitation from this cause is negligible, compared to the comfort given the baby by relieving his gastric distention. The air does not always all come up at once, often requiring several eructations. In

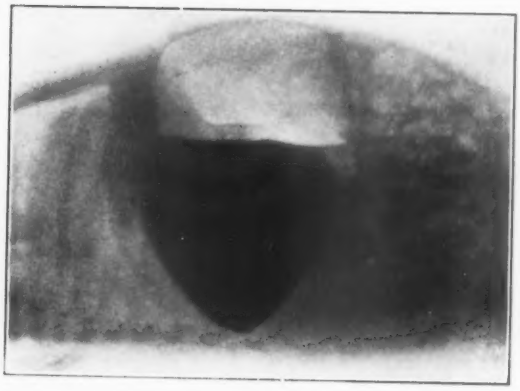

Figure 11

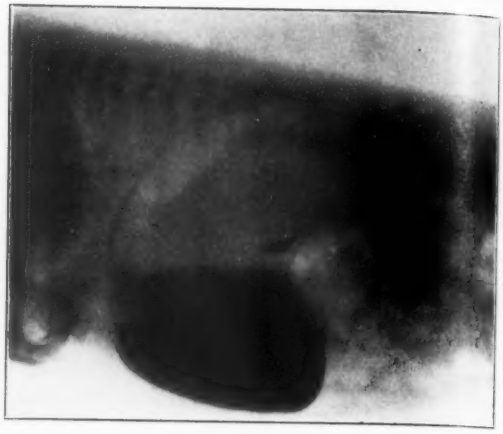

Figure 12

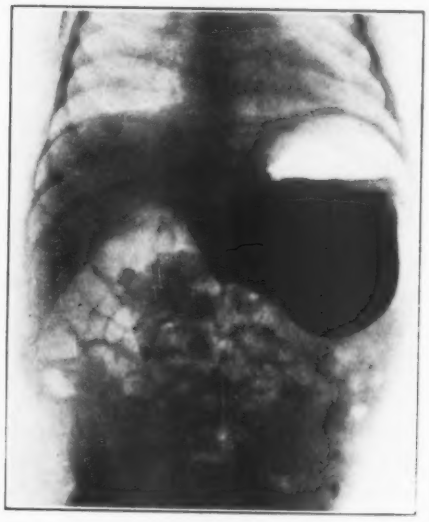

Figure 13

Figure 14

Figure 15

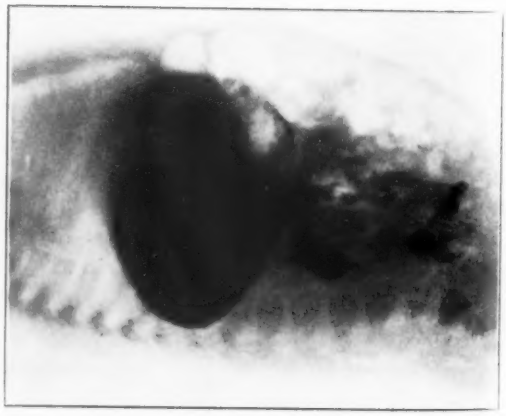

Figure 16

Fig. 11.—Healthy child, aged 6½ months; shown also in Figures 12, 13, 14, 15 and 16. Given usual feeding of 5½ ounces with bismuth subcarbonate ½ ounce. Taken well, no vomiting. Plate made two minutes after feeding was finished. Supine position. Right side against the plate. Enormous bubble of gas in stomach. Fluid line well above site of esophagus.

Fig. 12.—Three minutes after feeding. Prone positon. Left side against the plate. The milk has gravitated anteriorly, forcing the gas to the posterior part of the stomach. The position of the vertebrae show that the esophagus is now above the fluid line. The child was then held erect and eructated gas at once.

Fig. 13.—Four minutes after feeding. Erect position. Abdomen against the plate. The air bubble is at the upper part of the fundus, in a position whence escape is easy by the esophagus.

Fig. 14.—Five minutes after feeding. Prone position, the child's abdomen in contact with the plate. Note the gas only as a rim at the fundus end and the position of the pylorus compared with the preceding (Fig. 13). The child was again held upright and eructated more gas.

Fig. 15.—Eight minutes after feeding. Erect position. The gas bubble is still smaller in Figure 13.

Fig. 16.—Nine minutes after feeding. Supine position. Right side against the plate. There is only a very small bubble at the anterior part of the stomach. The stomach has contracted down on the milk. Compare Figure 11.

some cases (as in Figs. 1 to 6) a baby has to be held up every two to three minutes during the feeding to prevent vomiting from air swallowing. It is seldom possible to remove all the gas even when the baby eructates easily, but the small remaining amount causes no discomfort as a rule.

An illustrative case is that of the child from which Figures 1 to 6 were made. This was the second child, a large baby at birth. The labor was easy and short. She was breast-fed for two months, every two hours for twenty minutes. The milk then failed and the mother gave equal parts of milk and water with a "pinch" of sugar in each 5-ounce feeding, given every two hours by day during the third month. About 45 ounces were said to have been given in a day. The child was seen first at three months; weight 9 pounds 6 ounces. The nutrition was not very good, but the child was otherwise in fair condition except for a much-distended abdomen. She had vomited after nearly every feeding since birth, at times violently, but generally had simply regurgitated part of each feeding. The stools were three or four a day, yellow, soft, with occasional curds. She had some colic, and expelled a good deal of gas from the bowels. She slept well from 10 to 6, but the sleep was disturbed during the day. She had been fed in the horizontal position and had been put to bed in that position as a rule, but had occasionally been held up, and had eructated gas. A diagnosis was made of too frequent feeding, too large a total amount, air-swallowing and postural vomiting. The following formula was prescribed:

Milk, 4 per cent.....	18 ounces	367 calories	Fat.....2.4 per cent.
Lactose, 3 level tablespoonfuls		116 calories	Sugar.....6 per cent.
Water	12 ounces	483 calories	Protein.....2.1 per cent.
	30 ounces	Energy quotient.....52 per pound	

Fed 4¼ ounces every three hours; seven feedings.

The mother was carefully instructed to hold the child erect after each feeding in the routine manner (see summary).

One week later there was no gain in weight; still 9 pounds, 6 ounces. The child still vomited; the mother was discouraged and said the child was hungry. The milk was increased 2 ounces making the formula fat, 2.65 per cent., sugar, 6.3 per cent., protein, 2.4 per cent.; caloric value 554; energy quotient, 59 per pound.

One week later, the child still vomited a great deal, but had gained 14 ounces, weight 10 pounds, 4 ounces. The mother was then carefully questioned and said that the child vomited whether she was held up or left flat, but admitted she had not held the child up after every feeding, but only when the baby cried or seemed restless.

The whole purpose of the routine was again carefully explained and the mother was told to hold the child erect *before feeding, in the middle of the feeding and at the end of the feeding.*

One week later, vomiting was much less; weight 10 pounds, 10 ounces; same formula.

One week later, there was no more vomiting; weight 11 pounds, 2 ounces; same formula.

One week later, there was no more vomiting; weight 11 pounds, 14 ounces; same formula.

General comfort greater, sleep quiet, no colic. Energy quotient 50. The roentgenograms show that this child swallowed an excessive amount of air. During feedings it was possible to hear a gulping sound with each swallow.

Different nipples were tried, and the food was given slowly and rapidly without result. It is evident that this child swallowed as much air in taking 2 ounces of food as the average child does in taking a much larger amount, and therefore it was absolutely necessary that an opportunity be given to get rid of the air in the middle of the feeding, before the stomach became too greatly distended. We believe that this is an example of a large number of cases which are diagnosed as "nervous vomiting," "habitual vomiting" or even as pylorospasm.

Many very feeble babies vomit whenever put down on their backs even if held upright until the gas has all been expelled. The cardiac sphincter seems to have no power, and the milk simply flows out of their mouths whenever gravity has a chance to act. This may be stopped, in some cases at least, by constantly keeping the basket or bed at an angle of about 45 degrees, and never letting the child assume a horizontal position. The prone position may be borne, but it is better to have the head well raised.

The time taken at the bottle or breast is of importance. We believe that it is better for a child to take a feeding too rapidly than too slowly. Rapid nursing may choke the baby and cause coughing, gagging and vomiting if the pharynx fills too rapidly. Slow feeding, that is, twenty to thirty minutes, only multiplies the number of swallowing acts, diminishing the amount of milk and increasing the amount of air taken in each swallow. The old argument against rapid feeding is probably based on cases in which an enormous amount of milk was obtained from very full breasts and regurgitated from *over*feeding, not from *rapid* feeding. If an infant is weighed every minute while nursing a full breast it is often found that 50 per cent. of the milk is taken in the first two to three minutes, 75 per cent. in the first five minutes and after that the amount per minute decreases rapidly. Such infants show no more signs of discomfort or indigestion and do not vomit more than others, unless they stay at an empty breast sucking and swallowing air. A case may be cited in which a 6-weeks baby obtained from 2½ to 3 ounces in forty seconds and showed no signs of discomfort. The mother had to use a watch to time the feedings. If nursed one minute he vomited from overfeeding. Rapid feeding, therefore, in itself causes no symptoms, and except in the case of feeble infants there is no good reason why a feeding should not be taken in five to ten minutes, or at most in fifteen rather than the traditional twenty.

This is quite contrary to the usual teaching. Nurses are taught that the baby must spend twenty minutes at a bottle. Experience shows that the holes in the nipple must be made very small indeed to prevent a strong child from emptying a bottle in ten minutes. It is much better to have four or five large holes so that the milk runs freely without shaking the bottle when it is inverted, and let the baby regulate the

Figure 17

Figure 18

Figure 19

Figure 20

Fig. 17.—Healthy child, aged 7 months; shown also in Figures 18, 19 and 20. More or less vomiting since birth. Given usual feeding with bismuth subcarbonate ½ ounce. Feeding taken readily in fifteen minutes. Child kept in horizontal position. No vomiting or apparent discomfort. Five minutes after feeding. Supine position. Left side against plate. There is a large bubble of gas in the anterior part of the stomach. The milk lies in the posterior position. The small intestines contain a good deal of gas.

Fig. 18.—Eight minutes after feeding. Erect position. Abdomen against plate. As soon as this child was held erect the gas came up as a loud eructation. There is practically no gas left in the stomach, which has contracted down on the food. A little bismuth has entered the small intestine.

Fig. 19.—Seventeen minutes after feeding. Position prone — anterior abdominal wall against the plate. The difference in shape of the stomach between this and Figure 18 is striking. The pylorus has moved well over to the right, probably due to pressure on the abdomen by the surface upon which the child was lying. A good deal of bismuth has entered the small intestine.

Fig. 20.—Five hours, twenty-five minutes after feeding. Erect position. There is considerable bismuth still in the stomach, due to short interval feeding. The rest of the bismuth is in the cecum except for a small amount in the intestine, which contains little gas. The gas which was in the small intestine is now in the colon.

time for himself. Puny infants often become so discouraged at sucking at a nipple with the usual small holes that they will not try to take the bottle at all.

That posture is not without importance in abnormal conditions was shown by a case of pyloric stenosis. This child had a typical history of pyloric obstruction beginning at about six weeks, and had developed a large dilated stomach with tumor, peristaltic wave, projectile vomiting and wasting. The mother refused operation and even hospital treatment. With lavage and careful feeding — at first breast-feeding, then mixed breast and bottle — the child slowly improved, stopped vomiting and began to gain weight. At the time when the Roentgen-ray plates were made the child was 3 months old and gaining slowly. The bottle was not well taken at this time, so only about 2 ounces of feeding with bismuth subcarbonate could be forced down. There was still retention of considerable food in the stomach, and the two ounces of bottle made the child very uncomfortable. The first plate (Fig. 21) was made in the supine position. It shows the great dilatation and the double pouch-shaped stomach. The lower pouch is the pyloric end. The mother wanted to hold the baby upright at once, for she had been carefully taught the importance of so doing. During the time needed to take the first plate the mother became very anxious and begged that the child be held erect. She said: "I know he will vomit, and he has not vomited in two weeks, and I hate to spoil his record."

Her fears were realized, for the child did vomit violently — in a typically projectile manner, just as he had done at the onset of his symptoms. He was held upright immediately after the exposure and at once eructated a large amount of gas. This gas was not only swallowed air, but also the product of gastric fermentation due to stagnation. The residue in this child's stomach resembled kumyss. The child was held erect until the next plate was made (Fig. 22). This was twenty-five minutes later, and during that time he had eructated several times. Figure 22 shows only a small amount of gas in the stomach. The pyloric pouch has contracted down on the milk and a moderate amount of bismuth has entered the intestine. The child was held erect several times before the third exposure and again eructated. In Figure 23 there is almost no gas in the stomach. The intestines are almost free from gas in all the plates of this child. This is the more remarkable since there was known gas production in the stomach. The mother's care to allow the gas to escape from the stomach by the mouth not only was an important factor in stopping the vomiting, but also saved the intestines the necessity of passing the large volume of gas, with resulting colic and distention.

The teaching in practically all training schools for nurses has been that the baby should be kept in the horizontal position after nursing and must on no account be held up, or "moved about." This theory probably had its origin in an effort to counteract the trotting, rocking, jouncing habits of so many mothers — which of course may cause vomiting — the milk being splashed out of the stomach as it would be from a bottle. This does not take place easily, however, unless the child is held horizontally during the trotting or rocking. A careful census has been kept of all trained nurses in charge of infants in the practice of one of us in the last seven years, and only four or five have been found who had evolved the plan of holding the baby erect *as a routine*. A few more have done it at times when the child seemed to have colic or discomfort. Only one nurse has been found who was taught in her training-school that every child should be held up after every feeding.

Nurses train mothers nowadays. A young inexperienced mother takes the word of the trained nurse as law, and when doctor and nurse disagree, the doctor must indeed possess unusual influence over the mother to have an even chance of having his instructions carried out. Small wonder then that most babies are put into their beds to squirm and cry and vomit until enough milk comes up to relieve the distended stomach. After the trained nurse has left, a mother will sometimes pick up the baby and hold him against her shoulder in a desperate attempt to soothe away the colic with a little cuddling and patting. This is done with a guilty conscience at disobeying the nurse's ironclad rule. When the gas comes up and the baby goes to sleep and stays asleep when put back to bed, the mother begins to think. It needs but few repetitions of the experience for the intelligent mother to draw the correct conclusions. Unfortunately, the courage to disobey the nurse's law and the ability to draw conclusions from observation and experiment are rarely combined in one young mother.

Among the poorer classes, where there is no adverse suggestion from the nurse, the baby is often held upright as a routine. It must be admitted that the grandmother is often right on this point, much as she is abused for her interference with modern training and discipline.

Hospital feeding cases are usually examples of the bad results of the horizontal position. In many hospitals the babies are fed in bed and allowed to lie without change of position day in and day out, except when taken up to be bathed or moved to another bed. It is a simple matter to note in practically any case in such a ward that the abdomen is distended, with a large area of gastric tympany. Holding the child erect for a moment will bring up a large bubble of gas, and the percussion note of the epigastrium changes at once. Besides the effect on

the stomach, and on regurgitation, etc., it must be bad for such babies to lie in one position all day. Dr. W. P. Northrup pointed out years ago that it was the lack of holding in the mother's arms which made so many of these children do badly.

Older children who sit up by themselves regulate this matter under ordinary circumstances, but in acute illness, as pneumonia, there may arise a condition of gastric distention due to the horizontal posture. Raising such patients to the erect posture occasionally aids in the eructation of gas and relieves the pressure on the heart and lungs. This is probably one reason why surgical patients do better in the semi-erect position than when the foot of the bed was raised, as was formerly the general practice.

Medical text-books do not mention the posture of the child after or between feedings as a cause of vomiting or indigestion. The matter is dismissed with the general statement that the child should not be moved about after feeding. Medical writers are also silent on this subject with a few exceptions.

Rosenstern,[4] in a clinical lecture in Finkelstein's clinic, notes the importance of posture. He says that the practice of mothers and nurses of holding infants up and allowing eructations during and after feeding is regarded by doctors as an evil and is not described in medical text-books, but is found in a few written by nurses. He quotes Wilhelmina Voelzter to the effect that children held up vomit less and sleep better. He also notes that often young children after drinking a while get too full and can hold no more, become uncomfortable and if forced they vomit. If held up after a few minutes they eructate, and often a large amount of air. They will then take more food. This is especially true of children fed on dilute milk mixtures, in the early months, and in hospitals where nurses have no time to hold babies up.

Usener says:

> Daily experience teaches us that nurslings who are held up by nurses one-half to one and one-half hours after feeding almost always eructate more or less gas. Many mothers and good nurses hold the baby up during or after feeding from the experience that they take the feeding better, vomit less and are more quiet if they have eructated.

Kilmer[5] advised holding babies upright for colic to let the bubble which caused the colic escape.

Tweddell[6] says that after feeding the baby may be picked up if restless, held against the shoulder and patted gently on the back for a minute or two, as a little wind on the stomach may be the cause of the restlessness.

4. Rosenstern: Deutsch. med. Wchnschr., Sept. 26, 1912, p. 1834.
5. Kilmer: Practical Care of the Baby, F. A. Davis Company, 1903.
6. Tweddell: How to Take Care of the Baby, Bobbs-Merrill, 1913.

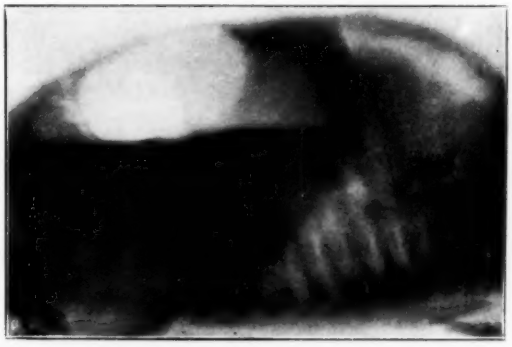

Figure 21

Fig. 21.—Child with pyloric stenosis, aged 3 months; shown also in Figures 22 and 23. Onset of symptoms at 6 weeks. Classical symptoms of stenosis or extreme spasm with gradual relaxation of spasm and improvement. Child doing well at this time, but stagnation and fermentation still taking place. Took bottle badly, only 2 ounces of food with bismuth subcarbonate 1:8. Five minutes after feeding. Supine position. Left side against the plate. Huge gas bubble anteriorly. Double pouch of stomach well shown. Child vomited violently just after exposure. Child was then held erect and eructated large amounts.

Figure 22

Figure 23

Fig. 22.—Twenty-five minutes after feeding. Erect position. Abdomen against the plate. There is only a small gas bubble at the cardiac end of the stomach. Considerable bismuth has entered the small intestine. The child was held upright several times during the next half hour and eructated more gas.

Fig. 23.—One hour and fifteen minutes after feeding. Erect position. There is very little gas left in the stomach. The intestines in Figures 22 and 23 show little or no gas. This is instructive in this case with known gas formation in the stomach.

Grulee says:

> Colic immediately after feeding is probably from stomach, later from intestinal gas. To expel the stomach gas the child can be held to the shoulder or across the lap.

CONCLUSIONS

Air is swallowed with the food by many if not by all infants.

The erect posture favors eructation of this air; the horizontal posture prevents it.

The horizontal posture, by preventing eructation, is an important cause of vomiting, colic, indigestion and disturbed sleep.

The following routine should be followed in feeding every infant:

Before feeding the infant should be held upright to allow the escape of any gas present in the stomach.

Immediately after feeding the infant should be again held up against the shoulder of the mother or nurse. He may be patted on the back or gentle pressure may be made on the epigastrium to encourage eructation of the swallowed air.

It may be necessary to interrupt the feeding one or more times to hold the child upright to eructate, in cases in which an excessive amount of air is swallowed.

After the gas is eructated the child should be put down to sleep, preferably in the prone position and with the head of the bed raised.

If restless he may be taken up after a short time to see if there is more air in the stomach.

Habitual tongue-suckers need to be held up several times between feedings, as they constantly swallow air. Other suckling habits must be prevented by mechanical restraint.

Feedings should be given at as long intervals as possible, depending on the gastric capacity and the total daily requirements.

A feeding should not be taken too slowly. From five to ten minutes are enough as a rule; fifteen minutes should be the maximum time at bottle or breast.

The importance of posture and the wrong teaching given to physicians and nurses in the past warrant the emphasis laid on so simple a matter.

257 West Seventy-Fourth Street—99 Claremont Avenue.

MEGACOLON AND MICROCOLON*

LANGLEY PORTER, M.D., AND ALANSON WEEKS, M.D.
SAN FRANCISCO

Pediatricians express difference of opinion as to the relative frequency of megacolon. Some authors of wide experience are able to make reports on only one or two such cases; that such experience is not general is attested by the fact that in the literature of the past four years there are more than one hundred papers each of which deals with one to six cases. It follows, therefore, that the affliction cannot be extremely rare. We have encountered six cases during the past five years; two of these passed from observation after one view and, therefore, are not included in this report. Microcolon, on the other hand, seems to be truly an unusual condition, as our search through the literature revealed but three certain and one doubtful case, although it seems probable that some reports under the caption "Stenosis of the Colon" may have referred to this condition.

The name "megacolon" has been used to describe the clinical picture characterized by obstinate constipation, great abdominal enlargement, visible intestinal peristalsis with the constitutional results of toxemia due to fecal retention. Constipation at times causes such retention of feces that fecal masses accumulate; these may shift and cause attacks of acute or subacute obstruction of the bowel. The literature referring to this congenital enlargement of the colon is ample. In his masterly article written in 1908, Finney presents a bibliography of 206 titles compiled by A. W. Fisher, complete to the time of his writing. We have searched the libraries diligently and find that over 100 papers on the subject have appeared since Finney's writing. One of the best of these later articles was written by Barrington-Ward and published in January, 1914. One must agree with Barrington-Ward's statement that Finney's paper so completely discusses all phases of the disease that it must remain the authoritative communication on this subject for a long time to come.

Although Finney shows that some twenty odd cases, one by Lewitt of Chicago (1867) and another by Jacobi, were clearly described before Hirschsprung published his studies of the malady in 1896, we owe the eminent Dane a debt for centering attention on the importance of the condition. This writer classified giant colon as: (*a*) true megacolon, first noticeable at birth or early infancy; (*b*) pseudomegacolon, making its first appearance in adult life and showing dilatation of the

* From the Medical Department of Leland Stanford, Jr., University.

colon with hypertrophy of its muscle, and arising secondary to some partial obstruction that came on during the postnatal period. Many writers deny pseudomegacolon pathologic validity, and, in fact, there seems to be little common to the two conditions.

Barrington-Ward divides the theories which seek to explain the origin of megacolon into four classes: (1) the mechanical; (2) the neuropathic; (3) the inflammatory and (4) the congenital. Under the mechanical theory he arranges five subheads: (*a*) the view of Barth, that an increased mesentery permits torsions of the sigmoid with partial occlusion and subsequent hypertrophy and gigantism of the intestine; (*b*) the view of Marfan, that the increased length of the intestine, especially the sigmoid portion, throws the bowel into a loop which causes kinking obstructions; (*c*) Treves' view, that the obstruction follows atresia of the anus and rectum; (*d*) Perth's idea, that valve formation is the cause of the obstruction; (*e*) Wilkie's conception, that the primary obstruction is due to the distention of the pelvic colon with meconium at time of birth or shortly after. As will be seen, none of these theories makes the difficulty of interpretation less.

Four different explanations are offered in elucidation of the neuropathic theory. It is suggested: (*a*) that there is a neuromuscular defect of the segment of the intestine; (*b*) that there may be a paralysis of a segment of the intestine; (*c*) that there is some implication of the sympathetic nervous system, and (*d*) that reflex spasm of the sphincter ani, a result of a fissure in ano, is the basic cause of the trouble.

Adherents to the third theory hold that a colitis that later becomes chronic originates those pathological processes which afterward produce the full picture of megacolon.

The proponents of the fourth theory assign the only cause which seems to explain the known facts. They hold with Hirschsprung that the condition is an anomaly of development and that the hypertrophy and dilatation both occur in the fetal intestine before birth. Concetti, while acknowledging the congenital origin of the dilatation, considers that the hypertrophy follows effort made to overcome the obstruction due to an inert segment of the dilated bowel. Other writers, among them Fenwick, believe that the hypertrophy is the congenital feature, and that the dilatation is the result of the pressure of the retained gases. In favor of the theory of congenital origin are age at onset, presence of constipation and other symptoms from birth as illustrated in all the cases here reported as well as by a study of the pathology. Furthermore, cases have been reported in which fully developed megacolon occurred in prematurely born children.

Barrington-Ward points out that developmental errors are most common at the junction of the proctodeum and hindgut and that the site of next greatest frequency is at the junction of the foregut and midgut. He considers that in megacolon the developmental error occurs at the former site, and he draws attention to pyloric stenosis as a possible example of developmental error in the later position. It is well known that the increase in the length of the intestine during the last month of intra-uterine life is usually about 25 per cent., but that there is great individual variation in this respect. At birth the infant's intestine is at least one-tenth longer in proportion to the body length than is the adult's. Much of this increase goes to make the sigmoid of excessive length in the infant. In the normal baby, growth rapidly balances this relationship, but in cases of giant colon such balancing is impossible, at least in well-developed cases. From such facts Finney infers that the colon is still under morphogenetic evolution, that the inconstancy of its length and minor details are but variations in the progressive evolution toward the final arrangement, and that the anomalies are but reversion types. According to him, in megacolon all the segments of the intestine may be involved, but more often the condition concerns that segment whose evolution is most primitive. He lays great stress on the thickness of the mesocolon in one of his cases. This he states to have been the result of enlarged lymph-glands together with enormously dilated lymph and blood vessels, which corresponded exactly with the gigantic section of the bowel. This appearance suggests to him a state of hypernutrition and he calls attention to the fact that this same increase in vessels is a feature of that gigantism which occurs in the limbs; he suggests that the conditions in the bowel may be parallel. A reference to the photomicrographs will show that in our cases very large much-dilated vessels occurred not only in megacolon, but in pyloric stenosis and in giant small intestine as well. But none of our material showed the lymph-node hyperplasia that Finney found in his case.

Briefly stated, Barrington-Ward's theory, which seems the most explanatory, is that there is a congenital abnormality in the lower end of the hindgut which takes the form of a muscular hyperplasia. It usually involves the rectum and spreads a varying distance up the pelvic and iliac colon. It may be that the whole of the hindgut is affected, but in some cases there is a definite localization of the change and the hyperplasia affects only a segment of the intestine. The hyperplasia of the muscles gives rise both to thickening of the walls and to increase of the bowel lumen. The physiologic functions of the intestine are in abeyance, and blocking is produced in the bowel behind, for a varying distance, which depends on the extent and duration of the

dilatation and hypertrophy. In time this fecal stasis produces necrotic changes in the mucosa, an ulcerative colitis follows which is the cause of death in most instances. It seems reasonable to us that Barrington-Ward and Finney each contribute facts that, taken together, explain the underlying causes of the pathology in these conditions.

CASES OF MEGACOLON

The following cases of congenital enlargement of the intestine are reported because they serve, some to throw light on the disputed points, and others to illustrate the danger of temporizing management, while the last case shows the efficacy of operative interference. The case of microcolon is included not only because of its rarity, but because it illustrates a condition opposite to megacolon and at the same time furnishes us with an abnormality in the small intestine which in every way parallels the pathological changes found in the large intestine in cases of giant colon. We present for comparison the photomicrographs of the giant colon, of the hypertrophied small intestine and of a case of hypertrophic pyloric stenosis. A comparative study of this picture will show how nearly identical the muscle and blood-vessel changes are in the three conditions. Our cases of megacolon are as follows:

CASE 1.—Baby S., referred by Dr. A. B. Spalding. Seen on the fourth day of life. The mother and father were healthy people. The birth normal, at full term. The child was brought in because of increasing abdominal distention and because the bowels had not moved since just after birth, when a small amount of mucus and meconium-like material was passed. No one had noticed the passage of any flatus.

On admission, the child weighed 8 pounds. It appeared somewhat dried-out, the skin hanging loose and lusterless. The child was well formed with no deformities except that the abdomen was exceedingly prominent. This enlargement was especially marked when contrasted with the dried-out limbs, and was taken to be due to gas retention. It was noted that there was an area of visible intestinal peristalsis which passed across the abdomen from the left iliac fossa to the border of the ribs on the right side. This area was horse-shoe-shaped and seemed to be due to movements in two separate segments of the intestine. The upper segment showed movement from left to right, while the movements in the lower segment appeared to pass from right to left. On examination the heart and lungs were normal; no tumors were determined in the abdomen. The abdomen was distended but not hard and was easily palpated. The anus was patent; the little finger entered an apparently normal rectum but was arrested just above the brim of the pelvis by what felt like a narrow band.

The child retained milk from one-half to one hour and then rejected it all. Attempts to give enemas were unsuccessful; the water returned uncolored. A bismuth meal was given and the child radiographed by Dr. Boardman. In the picture the stomach appeared normal, but the plates taken one and two hours after the meal showed no retention of bismuth in either stomach or intestine; all had been vomited. The child was kept alive by hyperdermoclyses of 4.5 per cent. glucose in normal salt solution.

Based on the finding in the rectum of a definite narrowing and of an apparent obstruction to the exit of the bismuth meal from the stomach together

with visible peristalsis, a diagnosis was made of multiple stenosis of the intestinal tract with narrowing at the duodenum in the small intestine and at the pelvic colon.

A surgeon was asked to see the child and to attempt multiple anastomosis. When the abdomen was opened the true state of affairs was revealed. A giant colon bulged into the wound and was returnable with great difficulty. The area of peristalsis proved to have been produced by a long redundant loop of the sigmoid which stretched across the abdomen. The outgoing loop crossed the transverse colon as high as the pylorus; it then wound around the lower

Figs. 1 and 2.—Patient 3, three months before death from pertussis.

border of the liver, and the incoming loop, starting above the hepatic flexure, ran thence to the left iliac fossa, where it terminated in a distinctly narrow but slightly hypertrophied area about 1½ inches in length. The child succumbed to the operation. Necropsy confirmed the findings revealed at the operation.

Unfortunately, the removed intestine was lost, hence it is impossible to report on the microscopic conditions present in this specimen.

This case is of interest in that it shows true giant colon to be a condition that may present itself at birth with fully developed dilatation and hypertrophy — this in spite of the view of those authors who

hold the dilatation to be variable and secondary, and others who have a like opinion about the hypertrophy. It is further of interest because, had the diagnosis been made and the intestine promptly opened for drainage, the child might have survived, and a later attempt could have been made to remove the large intestine, as has been done by Arbuthnot Lane. The way in which young children respond to gastro-enterostomy for pyloric stenosis is warrant for the faith that this child might have survived operative interference.

CASE 2.—A. H., 18 months of age. Seen in the course of private practice with the history of having had obstinate constipation and "big stomach" since the fourth month. He was the first and only child, born at full term, after protracted labor. The child had been difficult to feed in that he had not

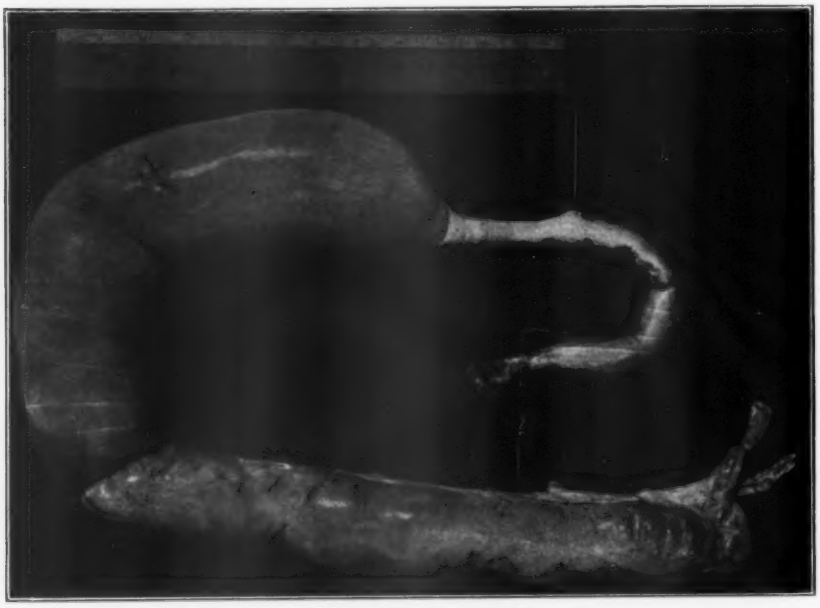

Fig. 3.—Colon from Case 3.

thriven on varying formulas and was astoundingly constipated. On one occasion, during the sixth month, he had passed several days (the mother said ten), without evacuation, and on many occasions there were periods of from two to five days without bowel movements. At such times the abdomen became tremendously enlarged beyond its ordinary huge size and the child would suffer attacks with rapid, shallow pulse, embarrassed breathing, and threatened collapse. The mother had learned that by the aid of a soft rubber catheter she could manipulate the contents of the bowel and so greatly relieve the child of the accumulated gas, and with this maneuver she carried the patient through many such crises. She had also discovered that the blocking of the bowel by feces was relieved and the gas most readily passed when the child's hips were held high above the shoulders. It is possible that in this position the retained mass was freed from the narrowed sigmoid by the aid of gravity.

On examination there was revealed an emaciated child with a huge abdomen, measuring twenty-four inches at the umbilicus, and looking, as one colleague

remarked, like a balloon tied to a little skeleton. The skin was dry and hung in folds everywhere except from the abdomen. The distention of the abdomen was exaggerated at the flanks, where the bulging was extraordinary. The diaphragm was high, and the costal region very wide. No visible peristalsis was noted. Masses, hard but doughy, were felt in the abdomen, one the size of a large orange on the right above the navel, another in the left iliac region about the same size, and smaller ones lay in what seemed to be the line of the transverse colon. These were moved about and felt with difficulty because of the extreme distention. Rectal examination revealed a wide, lower rectum, the little finger was stopped at about the pelvic rim by what seemed a definite

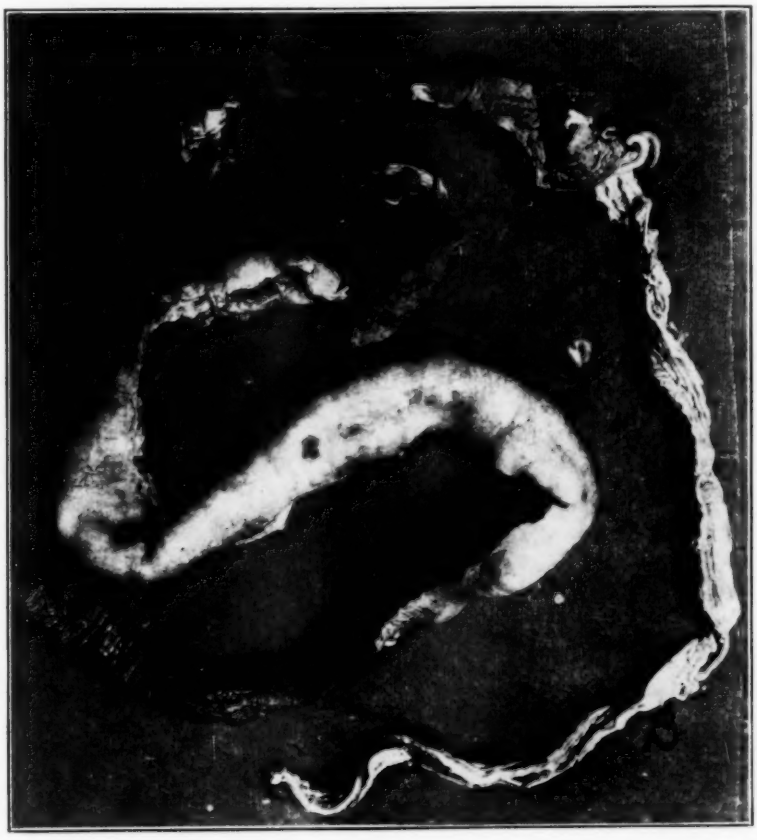

Fig. 4.—Intestines from case of microcolon. *A*, gigantism of small intestine; *B*, microcolon.

narrowing. Bimanual palpitation defined a tumor in the left iliac region, but pressure could not force it downward through the constriction.

The child now entered the Lane Hospital and it was decided to wait twenty-four hours and then to create an artificial anus. The patient seemed in excellent condition. Glycerin and water enemas were ordered and an attempt was made to produce evacuation. Several such enemas were given without result in the course of twelve hours and each time all of the fluid was returned unstained, according to the nurse's report. Apparently, however, a mass of

feces in the transverse colon was dislodged and dropped down into the narrow part of the intestine, where it produced an acute obstruction. At about 10 p. m. on the day of the child's entrance into the hospital, one of us was summoned hurriedly to find the child *in extremis* with the pulse running shallow and uncountable, the face gray, the lips blue, and the patient barely conscious. There was the most tremendous distention conceivable. In the absence of Dr. Weeks from San Francisco, a hurried summons was sent to another surgeon, but before he could reach the operating-room the child was dead. Obviously, the thing which should have been done was to incise, draw out the intestine and open. In the absence of surgical experience, and in the hope of finding skilled aid at once, one unskilled in surgery hesitated to assume this responsibility, but, undoubtedly, if such an emergency were faced again, the opening would be made without hesitation.

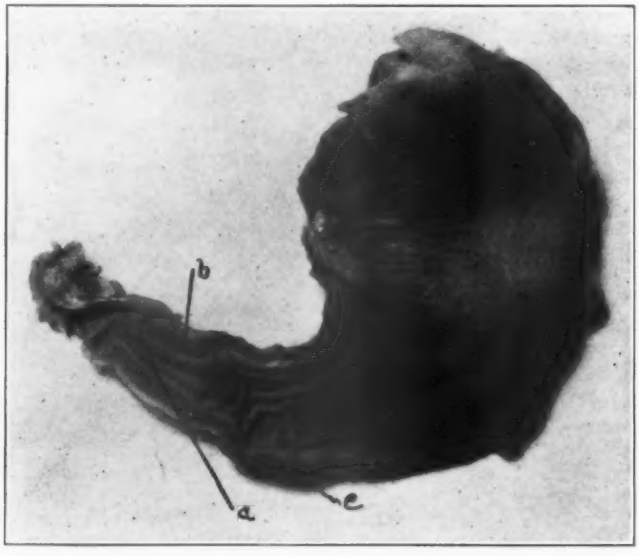

Fig. 5.—Hypertrophic pyloric stenosis, from photograph, full size; *a*, narrowing of lumen; *b*, ring muscle; *c*, hypertrophy of circular muscular coat of stomach. The narrow lumen of the pylorus is seen to be almost completely occluded at *a*. The wall of the pylorus is seen to be greatly thickened at *b*, and is continuous with the hypertrophied musculature of pyloric antrum.

Necropsy was denied us in this instance, so our only help from it lies in the illustration of the potential dangers of the condition and in the emphasis it puts on the view that the only safe procedure in these cases is to make a preliminary colotomy with artificial anus at the earliest possible moment, so that the intestine may be emptied of its retained material. After such procedure the poisoning ceases, the child's nutrition improves, and, with a wait of from two to four months, the physical conditions are so mended that there is warrant for a resection of the entire large intestine with the hope of permanent cure.

CASE 3.—E. S., male, 19 months of age, weight 15 pounds and 4 ounces, seen in consultation with Dr. Dudley Smith (Figs. 1 and 2). Full term, normal birth, birth weight 7¾ pounds. The abdomen was so distended at birth that the parents thought the child's hips were deformed because of the great contrast. Constipation at that time was very marked, and there was no evacuation until the fourth day of life, when scybalous masses of hardened meconium were passed, but in small quantity. In the second month a period of nine days elapsed without any movement of the bowel; the abdomen was greatly distended, and the child was much distressed. Evacuation was finally accomplished by enemas and castor oil, but these agencies produced only small marble-like scybala. At the time of this attack no signs of collapse supervened with the distention. The physician then in charge considered the condition to be due to unsuitable food, and the child was subjected to numerous changes in diet without effect. He gained slowly in weight and strength, but such attacks as just described recurred almost every month. On one occasion in the fourth month there was marked diarrhea which lasted for a week and was accompanied

Fig. 6.—High-power photomicrograph of the hypertrophied ileum. The normal mucosa is seen at *a*, under which lies the somewhat thickened submucosa. The muscular layers (*b* and *c*) are hypertrophied, especially the internal coat. The serosa, lying external to the muscular layers (*c*) is edematous.

by depression and great distress, which was relieved by castor oil and turpentine enemas. At six months, after obstruction of four days' duration, a characteristic attack supervened with pain, distention and collapse; at the eighth month another similar attack came on.

Dr. Dudley Smith now saw the child for the first time. He was able to effect evacuation of the bowels and to keep the child in fair condition by large doses of magnesia and the passage of a soft rubber catheter, which effectively relieved the distention. The child's nutrition began shortly to improve; his weight curve commenced to ascend and he reached 19¼ pounds in the seventeenth month, in spite of the fact that severe obstructive attacks occurred during the fourteenth and sixteenth months. During the eighteenth month the boy's weight fell during such an attack to 15¾ pounds, when one of us saw him for the first time and we coincided with Dr. Smith's opinion that oper-

ative interference held the only promise of relief for the condition. This help was not to be, however, as the child was even then in the first stage of pertussis, to which he later succumbed. The necropsy revealed a typical megacolon (Fig. 3).

The physical examination of this child showed simply a distended abdomen in a rickety subject who exhibited visible peristalsis over the middle and upper section of the abdomen; there were palpable fecal masses, and the rectum on examination revealed a narrowing at the pelvic brim. This case, like the last, shows the method of obstruction that occurs in this pathologic state and in each instance, the pain, collapse, and obstruction were relieved after the passage of a hard, narrow fecal mass which apparently had lodged in the narrowed sigmoid.

Fig. 7.—Photomicrograph of hypertrophied wall of pyloric antrum. The mucosa (*a*) is normal and is covered with a layer of mucus. Beneath it lies ths submucosa, which is loosened from the underlying internal muscular coat (*b*), which shows a marked hypertrophy. Throughout it are seen fibrous connective tissue strands extending down from the submucosa. The external muscular layer (*c*) is not much thickened.

Case 4.—E. P., male, 22 months of age, referred by Dr. Hughes of Sonoma, Cal., with the diagnosis of Hirschsprung's disease. This was the second child, full term, normal birth. Parents and other children normal and in good health. Symptoms were not noticed by the parents until the fourth month of life. From that time the history was like that of the other cases reported, malnutrition and difficult digestion with attacks of obstruction and collapse. On examination we were confronted with a yellowish gray-skinned anemic child, weighing 22 pounds. There was an enormous abdomen, which measured thirty inches at the level of the umbilicus. We saw no visible peristalsis on this occasion, and there was no such tenseness of the abdomen as had been met in the other cases. Palpation of the abdomen revealed several tumors; the largest, the size of a fetal head, lay in the right iliac fossa, extending upward and toward the right where the bulging was marked. Other similar masses lay above the umbilicus and to the left of it. The largest mass was

smooth, easily movable and was taken to consist of feces lying in the caput coli. Rectal examination revealed no narrowing; on the contrary, the rectum and colon were very large and roomy. Previously, a diagnosis of tuberculous mesenteric adenitis had been made. Bimanual examination with the finger in the rectum revealed a smooth, velvety peritoneum and contradicted this assumption.

Dr. Weeks now saw the child for the first time and advised operation, proposing a two-stage procedure. At the first operation the abdomen was opened on the left side and an artificial anus was created. The method of Levison of San Francisco was utilized and the retained fecal masses were then evacuated and we found that the one which had been discovered on the right side was lying, not in the caput coli, but in the redundant loop of the huge sigmoid. This giant sigmoid crossed over the abdomen and filled most of it; it overlay the apparently normal caput coli and thus gave rise to the erroneous impression. The operator drew the intestine down as far as possible into the left iliac fossa, where it was fastened, opened, and its contents evacuated. The child did well, the toxic symptoms manifested in the dry, yellow skin, anemia,

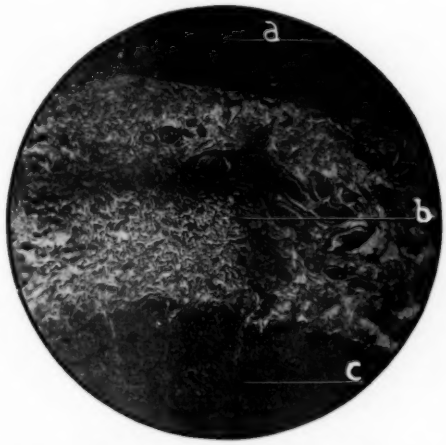

Fig. 8.—High power photomicrograph of megacolon. The mucosa at (*a*) is normal. The submucosa (*b*) is markedly thickened and fibrous and contains many large dilated vessels filled with blood. The muscle layer (*c*) is also thicker than normal.

and poor nutrition gave way, and at the end of six weeks the child had gained 12 pounds in weight. For four months evacuation was through the artificial opening, then as the child had made such great improvement, it was decided to resect the intestine. This was done at the Children's Hospital in San Francisco. Under ether the redundant sigmoid and the intestine above it up to what appeared to be the normal first part of the transverse colon were resected. After removal this redundant loop was 15 inches in length and in diameter about 3 inches, and its walls about 5/16 of an inch in thickness. The freed upper end was stitched into the severed end of the rectum; this was accomplished with great difficulty because the rectal tissues were thick from hypertrophy and the tube at that point was nearly three times as wide as was the implanted end of the large intestine.

The report of the microscopic section (Figs. 10 and 11) by Jean Oliver is as follows: "The sections show a normal mucous membrane with well-marked lymphoid follicles which are somewhat hyperplastic. All the muscular elements of the intestine are markedly hypertrophied, including the muscularis mucosae.

The submucosa is rather fibrous and the submucosa and serosa normal. Many large dilated blood-vessels filled with blood are found."

The anastomosis was successful except that there was a small area of imperfect union which gave rise to a slight fecal fistula. This opening, however, closed spontaneously after about two weeks and the patient went on to apparent recovery with occasional attacks of retention of feces and moderate

Fig. 9.

Fig. 10.

Figs. 9 and 10.—Low-power photomicrographs of the microcolon (Fig. 10) and the dilated and hypertrophied ileum (Fig. 9) showing the relative size of the gut of the different levels and the thickness of its walls. The marked dilatation of the ileum (Fig. 9) masks to a certain degree the hypertrophy of the muscular coats so that they do not appear much thicker than those of the microcolon (Fig. 10).

abdominal distention, which were apparently due to the fact that the huge rectum was deficient in tone and expulsive power. For some months the improvement was marked and progressive, but at no time has the patient done so well as during the period while he carried a freely opened artificial anus.

The bowels have to be evacuated by the aid of small low enemas of glycerin or soap and water. This dislodges what appears to be a normal fecal mass from the rectum and each enema is followed by a normal-appearing evacuation.

Six months after the operation the mother stated by letter that some distention was recurring. This is undoubtedly due to the fact that the portion of transverse colon which was left suffered from the same pathologic defect as the portion amputated, although probably to a less degree, and this region is now giving way under the stress of pressure. It seems probable that the remaining colon will yet have to be removed.

CASE OF MICROCOLON WITH HYPERTROPHIED SMALL INTESTINE

During the period of our interest in megacolon Baby R., male, 5 days of age, was brought into the Lane Hospital *in extremis,* deeply jaundiced and cyanotic, incessantly vomiting, ejecting meconium-like material. The child had had no evacuation since birth, had taken but little food and had passed urine a few drops at a time only. The vomiting had persisted for forty-eight hours before entrance. The abdomen was distended and tense; it was possible to diag-

Fig. 11.—High-power photomicrograph of microcolon. The mucosa (*a*) is normal, and the underlying submucosa is somewhat fibrous. The muscular layers (*b* and *c*) are relatively thicker than normal, but the decrease in the size of the intestine is evident in all its layers, no single layer being decreased at the expense of the others.

nose the presence of free fluid in the peritoneal cavity. The anal orifice was normal and the little finger moved freely into the rectum and was stopped just above the pelvic brim by a marked narrowing. A diagnosis of intestinal obstruction due to stenosis at the sigmoid was made, but the child was past aid and died an hour after admission. The necropsy was performed by Dr. William Ophüls, whose notes are as follows:

"Strongly built, male baby, 54 cm. in length. Cyanosis. Anus well formed, patulous. No obstruction in any of the natural orifices. Skin shows yellowish hue all over.

"Some gas in peritoneum and about 350 c.c. of distinctly fecal fluid. Peritoneum is inflamed and covered with soft greenish shreds of fibrin. Omentum is small, hyperemic, contains no fat. Transverse and descending colon very thin and contracted. Sigmoid flexure is pulled up out of pelvis to a certain extent and fastened by vascular adhesions to the mesentery of one of the loops of small intestine. These adhesions are below the point of greatest distention of the bowel. About 10 cm. above the ileocecal valve is a small perforation.

"Upper part of small bowel distended with gas, lower part very much distended; diameter of intestine below is 3.5 cm. in places. Further down distention decreases, increases again, and at the ileocecal valve the intestine has normal diameter.

"Above-described adhesion is below the point of greatest distention of bowel; about 10 cm. above the ileocecal valve is an oval perforation, about 5 mm. in diameter, situated close to the mesenteric attachment, through which fecal matter escapes.

"Upper part of the small bowel is filled with soft greenish brown fecal material. In lowest part of small bowel is thick, pasty material. The ileocecal valve is open. The large intestine is very much contracted. It is almost empty but in some places there is a small amount of dried-up mucus. The rectum is of normal caliber, its mucous membrane hyperemic, with some blood in rectum. Appendix is normal. In region of perforation mucous membrane shows marked hyperemia and extensive hemorrhages, and there is some evidence of necrosis of the mucous membrane. Very much distended part of lower small intestine shows well-marked hypertrophy of muscular wall, being almost 4 mm. thick in places. Mucous membrane is distended, part much inflamed, superficial erosions in places. Necropsy findings revealed no other abnormalities."

Sections were made from the hypertrophied areas of the small intestine by Jean Oliver, whose report is here given.

"Section of the small intestine through its enlarged part shows marked hypertrophy of the muscular layers. This is especially prominent when one considers that there is also great dilatation, which tends to obscure the hypertrophy of the walls. The mucous membrane is extremely hyperemic and there are many large hemorrhages, especially in the submucosa. A considerable layer of mucus covers the epithelium. The serosa is also hyperemic and there is beginning cellular infiltration and deposit of fibrin on its surface. The blood-vessels are large, dilated and filled with blood.

"Sections of the microcolon show the same hyperemia and hemorrhages in the mucous membranes but to a less degree. The submucosa is quite fibrous and also shows considerable diffuse hemorrhage. The muscular layers appear thickened, probably owing to the contraction of the intestine. The subserosa shows marked cellular infiltration and there is a heavy deposit of fibrin in the serosa."

The case of microcolon is included in these reports, not only because of its striking contrast to the megacolon cases, but also because there was also present a condition of hypertrophy of the small intestine (Fig. 4) in which the changes in the muscle and fibrous tissues parallel in every pathologic way the changes of the analogous tissues in the large intestine found in the cases of Hirschsprung's disease. It always seemed to us that the innate pathology underlying megacolon is akin to, if not identical with, the changes in tissue found in hypertrophic pyloric stenosis, especially in that form wherein there is general hypertrophy of the circular muscle of the stomach wall as well as of the ring muscle at the pylorus (Fig. 5). This case of microcolon with gigantism of the small intestine provides a true example of the same marked growth of muscle and hypertrophy of fibrous tissue with gigantism of vessels occurring in the small intestine.

Microphotographs of the sections of the three conditions have been made in the standard Pathological Laboratory by Jean Oliver (Figs. 6,

7, 8, 9, 10 and 11). A study of them will reveal how like the muscle affection and the marked hypertrophy in the mucosa connective tissue and vessels is in the three conditions. The photograph of the stomach from a patient with pyloric stenosis also is evidence for this point of view.

The consideration of our cases of megacolon gives some evidence on disputed points. Certain writers have denied the congenital origin of this condition. Our Cases 1 and 3 exhibited symptoms at birth and in Case 1 a typically developed giant colon was revealed by operation and necropsy. Cases have been reported in which giant colon was discovered in prematurely born children.

It has been questioned whether the sigmoid was narrow in all cases, and it has been asked if the true origin of the dilatation and hypertrophy was not an obstruction that blocked the passage with feces and allowed the work of the intestine and the stress on it to be followed by increased production of muscle and ballooning from the retained gas. Case 4 of this series, in which the gigantism included every portion of the intestine to the anus, shows that the sigmoid narrowing is not present in every case. Case 2 is instructive in demonstrating the potential and emergent dangers to life of the condition, and with Case 4 causes us to resolve that in the future even the youngest child encountered with this trouble shall have the benefit of immediate colotomy and an artificial anus. The well-being of Patient 4 during the time he carried an artificial anus was so striking and the operation is so easy that these facts, taken with the well-known tolerance of infants to abdominal interference, make such an operative interference most reasonable, especially when we consider the unhappy course of Case 2 which demonstrated so completely the extreme danger of temporizing and of trying to evacuate the bowel through the rectum in those cases of megacolon with a narrow sigmoid.

As one considers the condition of gigantism in the intestine, the question presents itself: Are there mild cases of the disease? One who deals much with infants cannot but feel that such cases occur, and are not very unusual. There are obstinately constipated infants, who, while not rachitic, have abdomens that bulge inordinately and in whom the bulging for the most part follows the line of the colon and fills out the flanks to a marked degree. Such children are apt to be uncomfortable, to take food badly and to fail in weight, but they are not distressed by flatus. When their bowels are kept free all the symptoms disappear except the ballooning of the large intestine, and growth seems to adapt the children in time even to this visceral enlargement, for the individuals in this group seemed to do well once they are past infancy. The final proof of this contention must rest on

necropsy of such a child who has met death from another cause, as the condition itself is never fatal, nor indeed is it severe. Hertz has called attention to a class of infants in whom roentgenograms show a lengthened sigmoid, and it may very well be that such infants correspond in the group of megacolon to the cases of mild hypertrophy with spasm that we encounter in the pyloric stenosis group.

The proper feeding of infants with congenital enlargement of the colon is of importance and presents difficulties. High fat formulas are not adapted to this condition, as soap stools give more trouble in all classes of megacolon than do other types of stools. Giant colon cases seem to respond to high percentages of dextrin, yet care must be exercised, as fermentation and gas formation have to be discouraged. Enemas are on the whole the best means of producing bowel evacuations. Castor oil is to be shunned, as is magnesia. In older children milk should not be given.

The clinical picture of congenital enlargement of intestine and the colon is so characteristic that it seems hardly needful to discuss the differential diagnosis. The one case in our series which led to confusion was that of the new-born infant. The differentiation between multiple stenosis and megacolon in the new-born is certainly difficult, but the character of the peristalsis and the mildness of the digestive symptoms should have been a guide to a proper diagnosis. This case was rendered more difficult by the vomiting, which was extreme, and which probably is a most unusual accompaniment of congenital enlargement of the intestine. The only other condition which might possibly lead to a mistake in diagnosis is tuberculous peritonitis, and this mistake will never be made if the child is examined under an anesthetic by the bimanual method, a finger of one hand in the rectum and the other hand on the abdominal wall. The characteristic smooth feel of the healthy peritoneum instantly excludes the presence of tuberculous inflammation. The gristly fibrous bands and the rolled-up lower edge of the omentum in tuberculous peritonitis give to the examining finger a sensation that is absolutely pathognomonic. (In passing, it may be said that there is no maneuver in diagnostic examination of children of more value than this bimanual rectal examination if it be made while the child is under the influence of an anesthetic.) It is unlikely that any of the abdominal tumors will be confused and hardly possible that the dilatation due to chronic intestinal indigestion in rickety children will mislead one.

It is probable that megacolon is a more common deformity than is generally thought. The symptoms are present from birth. They are: an enlarged abdomen, visible intestinal peristalsis, obstinate constipation, later, anemia and toxemia from fecal retention, accompanied

sometimes by recurring attacks of intestinal obstruction. Pathologic hypertrophy with dilatation may include the entire intestine, or the dilatation may cease at the sigmoid, which will be hypertrophied, but with a narrow lumen. That isolated segments of the large intestine may be affected while most of the intestine remains intact and healthy is to be doubted. Fatal collapse can occur from fecal obstruction, and such obstruction may be brought on by injudicious attempts to empty the bowel. The one measure that promises relief is operative procedure, which may be utilized in either two or three stages: colotomy, with artificial anus (this cannot be done too early in the child's life or in the course of the case), later excision of the entire large intestine with anastomosis from the ileocecal region into the rectum; or anastomosis may be done as a second step and resection of the entire colon be made a third operation.

240 Stockton Street—350 Post Street.

A STUDY OF THE NUTRITIVE VALUE OF SOME PROPRIETARY INFANT FOODS

II. AS MILK MODIFIERS *

RUTH WHEELER, PH.D.
URBANA, ILL.

An earlier number of this journal contained a report[1] from this laboratory of a study of the nutritive value of four commercial infant foods, two containing milk and advertised as complete foods, and two made chiefly from grains and advertised as milk modifiers. The object of that investigation was "to study the nutritive value of the foods in themselves," "not cooked nor mixed with milk as such foods are commonly given to babies, but as nearly as possible as they come from the manufacturers, with only such modifications[2] as are necessary to adapt the food to the species of experimental animal chosen." The necessary modifications were made in all cases by the addition of purified casein and the salts of milk in the form of Osborne and Mendel's "protein-free" milk. The two foods made largely from milk sustained entirely normal growth in young animals and long continued maintenance in adults. The two foods made chiefly from grains and containing no milk did not sustain normal growth; one allowed maintenance, the other did not.

In the series of experiments now to be reported, the foods of this latter group, Mellin's Food and Eskay's Albuminized Food, were studied as milk-modifiers, whole fresh milk in varying amounts being mixed with each day's ration (4 gm.) of the food powder and fed to young animals. The object in view has been to find out *in how far* these foods satisfy the nutritive requirements of growth; whether or not they may furnish a significant proportion of the dietary of a growing animal. Various claims are made for the many milk-modifiers on the market. They are perhaps most commonly said (1) to dilute the excessive protein and salts content of cows' milk, and (2) to

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* From the Research Laboratory of the Department of Household Science, University of Illinois, Urbana, Ill.

1. Wheeler and Biester: A Study of the Nutritive Value of Some Proprietary Infant Foods, AM. JOUR. DIS. CHILD., March, 1914, 169.

2. Approximate composition of the mixtures fed: Protein, 18 per cent,; salts, 3.7 per cent.; carbohydrates, about 75 per cent.; fat 1 to 3 per cent.

increase the digestibility of milk by softening the curd. Either of these purposes could be served by small proportions of the food powder and might perhaps be as well served by modifiers much more economical than proprietary infant foods. If, on the other hand, these foods may safely form a significant part of the ration, it would be of value to know wherein they fail as complete foods; whether, for example, from the lack of essential accessory substances or from an inadequacy in the proteins contained in them. With these questions answered, the foods could be used to much greater advantage.

The experimental animals used were, as before, albino mice, and the period of their lives chosen for the feeding trials was that of most rapid growth, that is, from the close of the nursing period — the twenty-first day after birth — until after the mice reach sexual maturity at about the sixtieth day after birth, when the rate of growth markedly decreases. In spite of the facts, that the experimental animals used were not babies and that the observed period not the nursing period, it is hoped that the results of the experiments may prove suggestive to pediatrists; for while the nutritive requirements for growth vary decidedly with the species of animal so far as quantitative considerations are concerned, the food substances needed appear to be the same for all the higher animals. This is indicated by the fact that animals of various species have grown normally on an exclusive diet of cows' milk or of egg yolk. It appears that all animals must have certain amino-acid combinations, carbohydrates, salts and probably some accessory food substances; and a food mixture which will not support proper growth in one species of animal, when the amount of protein, salts, etc., corresponds with the proved nutritive requirements of that species, lacks some essential, and may well be studied with great care if it is to be fed as a significant part of the diet of a baby.

In regard to growth requirements during different periods of life, much the same arguments hold. Even quantitatively there is probably much less change in the optimum composition of food as the animal grows than is often supposed. Certainly the same food substances serve for real growth alike in the nursing period and later. The concentration of the food is a somewhat different matter and will be discussed later.

As before, the mice were caged in groups of two or three; white crepe paper napkins were used for bedding and were put into the cages clean every day; twice a week the cages were sterilized by boiling. Distilled water was supplied in inverted bottles. Food was given three times a day, between seven and eight in the morning, at four in the afternoon and between eight and nine in the evening. At first there

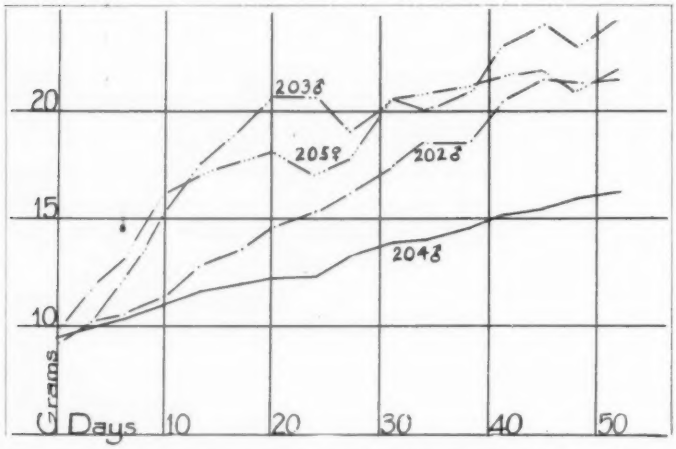

Chart 1.—Growth of four mice on Mellin's Food with varying amounts of milk added to the ration: Mouse 204 ♂ had Mellin's Food without milk; Mouse 202 ♂ had 1 c.c. of milk mixed with the day's ration; Mouse 203 ♂ had 3 c.c., and Mouse 205 ♀, 5 c.c. of milk. All had always an excess of food in the cage.

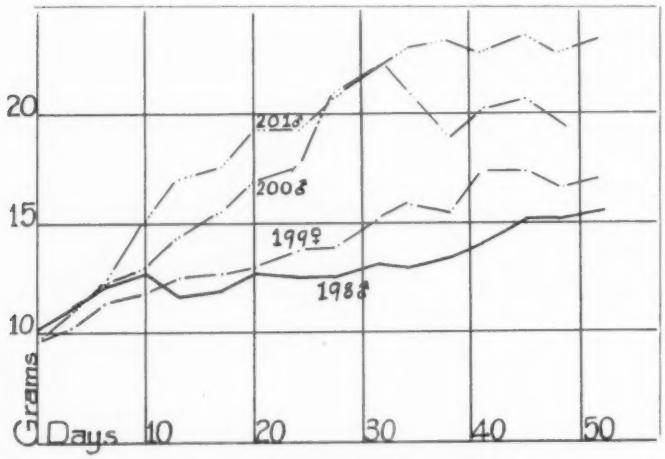

Chart 2.—Growth of four mice on Mellin's Food with varying proportions of milk added: Mouse 198 ♂ had no milk; Mouse 199 ♀ had 1 c.c.; Mouse 200 ♂ had 3 c.c., and Mouse 201 ♂, 5 c.c. of milk added to the day's ration. All had always an excess of food in the cage. The effect of 1 c.c. of milk is less evident than in the preceding chart because Mouse 199, a female, would normally grow more slowly than Mouse 198, a male.

was also a noon feeding, but as food given at this time was seldom touched, this feeding was omitted in the later series. Any mixture containing milk was kept on ice until put into the cages, and all containers were sterilized by boiling every time they were used.

Previous work had shown that the two food powders needed somewhat different treatment, Eskay's Food requiring the boiling recommended by the manufacturers. As far as the amount of milk allowed, both were fed as a thin paste as in the earlier work. When small proportions of milk were used, the food powder was mixed first with distilled water to a thick paste, then the milk was added from a pipette or a graduate and very thoroughly stirred in. With the larger proportions of milk, no water was used. Except with the largest amounts of milk, the physical consistency of the final mixtures was practically the same throughout and similar to that adopted in earlier work.

Both Mellin's Food and Eskay's Food had to be modified before they could be fed with small proportions of milk, both of them containing too little protein for mice, and Eskay's needing also an increased salt content. As in earlier work, the protein added was purified casein and the salt mixture, Osborne and Mendel's "protein-free milk." Both of these had been satisfactorily used before in long-continued experiments, both as the sole source of protein and of salts, respectively, and as modifiers of infant-foods, so that no unsatisfactory results can be attributed to them. With increasing proportions of milk, less added casein and protein-free milk were needed. Some of the mixtures contained unmodified foods mixed with the milk. Both the formulas for mixing and the composition of the final mixtures will be found in Tables 1 and 2.

TABLE 1.—FORMULAS AND COMPOSITION OF MELLIN'S FOOD MIXTURES

Formula—	1	2	3	4	5	6	7	8
Mellin's Food	93%	93%	93%	93%	93%	93%	90%	90%
Casein	7%	7%	7%	7%	7%	7%	7%	7%
Olive oil	0	0	0	0	0	0	3%	3%
Milk to 4 gm. food		1 c.c.	2 c.c.	3 c.c.	5 c.c.	10 c.c.		1 c.c.
COMPOSITION IN TERMS OF TOTAL SOLIDS								
Protein	18.3%	18.6%	19.2%	19.5%	19.8%	20.8%	17.7%	18.3%
Ash	3.6%	3.7%	3.7%	3.7%	3.7%	4.2%	3.5%	4.2%
Fat	0.7%	1.6%	2.5%	3.4%	4.8%	8.0%	3.6%	4.5%
Carbohydrate	77.4%	76.1%	74.6%	73.4%	71.7%	67.0%	73.2%	72.9%
Calories to 100 gm. (computed)	398	413	408	411	421	435	408	416

TABLE 2.—FORMULAS AND COMPOSITION OF ESKAY'S FOOD MIXTURE

Formula—	1	2	3	4	5	6
Eskay's Albuminized Food	67%	67%	73%	73%	100%	100%
Casein	13%	13%	11%	11%		
Protein-free milk	18%	18%	16%	16%		
Olive oil	2%	2%				
Milk to 4 gm. food	3 c.c.	5 c.c.	10 c.c.	20 c.c.	40 c.c.	50 c.c.

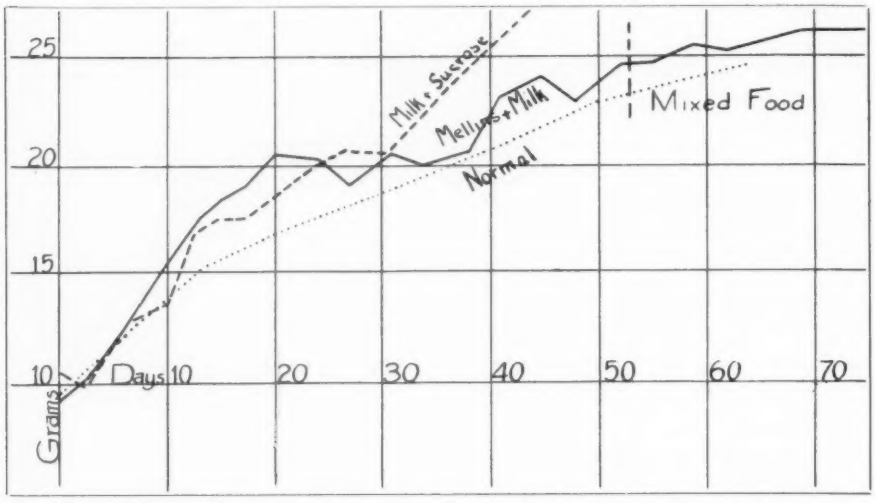

Chart 3.—Comparative growth of Mouse 203 ♂ (repeated from Chart 1) on Mellin's Food plus 2 c.c. of milk to the day's ration (4 gm.) and of Mouse 197 ♀ on milk plus 6.5 per cent. of sucrose. The dotted line indicates normal growth for mice.

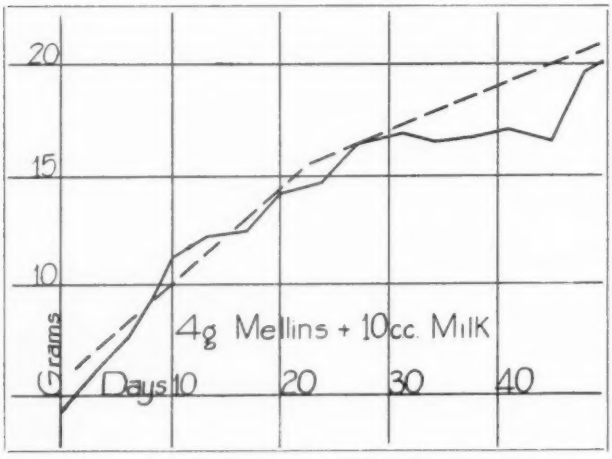

Chart 4.—Growth of Mouse 196 ♂ on Mellin's Food plus 10 c.c. of milk to the daily ration. The interrupted line shows normal growth.

COMPOSITION IN TERMS OF TOTAL SOLIDS

Formula—	1	2	3	4	5	6
Protein	19.2%	20.0%	18.5%	21.7%	18.7%	21.2%
Ash	3.8%	3.9%	3.8%	4.2%	3.8%	3.7%
Fat	5.3%	6.8%	5.1%	12.6%	16.9%	19.2%
Carbohydrate	71.7%	69.2%	72.7%	61.5%	60.7%	55.9%
Calories to 100 gm. (computed)	408	428	421	448	483	495

The effect of possible variations in the composition or in the bacterial content of the milk was watched by feeding, to at least one animal in each group, milk modified only by the addition of 6.5 per cent. granulated sugar. This mixture had a composition similar to that of the other foods. (See Table 3.)

TABLE 3.—COMPOSITION OF MILK AND SUCROSE MIXTURE

MILK + 6.5 PER CENT. SUCROSE. COMPOSITION IN TERMS OF TOTAL SOLIDS

	%
Protein	18.2
Ash	3.7
Fat	19.2
Carbohydrate	58.8
Calories to 100 gm. (computed)	508

MELLIN'S FOOD

In the earlier investigation, it was found that Mellin's Food unmodified could not serve as an exclusive ration either for young or for adult mice. The animals on such a diet steadily lost weight and eventually died unless the food was changed. Since other foods of closely similar gross composition served perfectly for maintenance through long periods, the difficulty could not lie in the percentage of protein, etc. It might, however, lie in either of two factors, namely, lack of essential accessory substances such as "vitamines" or "growth hormones," or in an inadequate supply from its proteins of essential amino-acids. Osborne and Mendel[3] have recently shown conclusively that lysin is necessary for growth, and in two of the chief proteins of the grains from which Mellin's Food is manufactured, hordein of barley and gliadin of wheat, lysin is absent or present only in traces. The addition of 1 c.c. fresh milk per day to the diet of each mouse on unmodified Mellin's[4] Food did not stop the decline in weight, so the trouble was not all in the lack of accessory substances. Five per cent. casein added to the ration, stopped the decline and allowed the lost weight to be regained as rapidly as on a general mixed diet;[5] but no

3. Osborne and Mendel: Jour. Biol. Chem., 1914, 325.
4. Wheeler and Biester: AM. JOUR. DIS. CHILD., March, 1914, Chart 19.
5. Wheeler and Biester: AM. JOUR. DIS. CHILD., March, 1914, Chart 18.

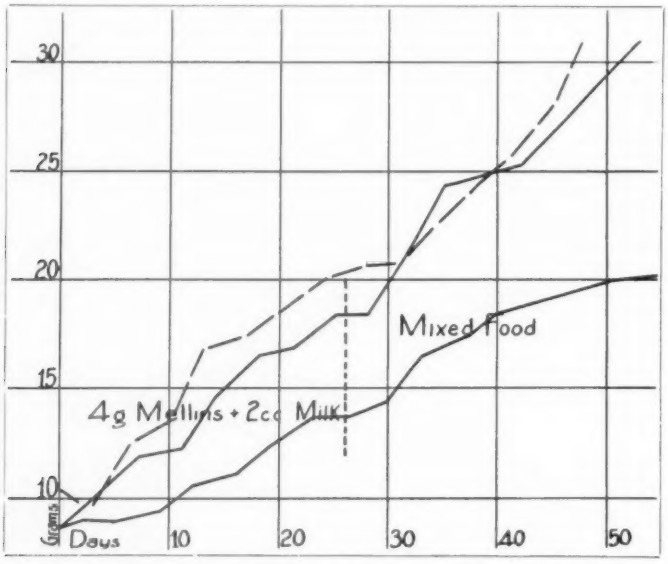

Chart 5.—Growth of two mice, Nos. 217 ♀ and 209 ♂ on a diet of Mellin's Food with 2 c.c. of milk added to each day's ration. For comparison the growth of a control animal 197 ♀ on milk plus 6.5 per cent. sucrose is shown in the interrupted line.

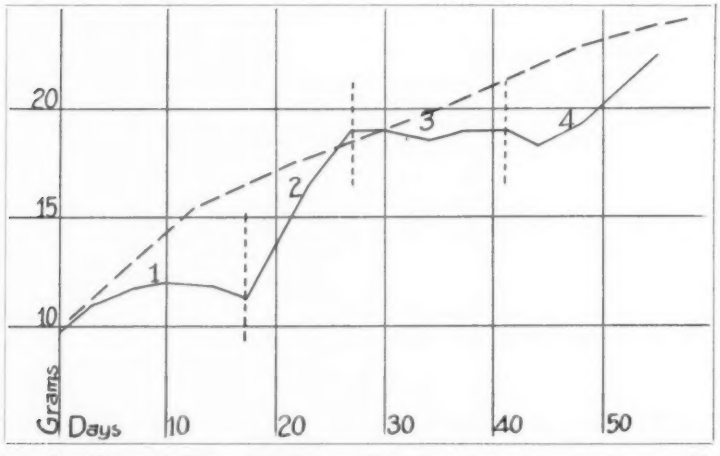

Chart 6.—Mouse 194 ♂, showing maintenance but no growth on a Mellin's Food-oil-casein food (periods 1 and 3) and rapid growth when to the day's ration of this food was added 1 c.c. of fresh milk. The interrupted line shows normal growth for mice. For the composition of the food, see Table 1, Formula 7.

further significant increase took place. There was practically no growth. The casein was able to supply the lack partially, but not completely.

In the present series of experiments a number of modifications of Mellin's Food were made. The composition of the most significant of them is given in Table 1, and some of the results of feeding them in Charts 1 to 6, inclusive. In each group of feeding trials, a single litter of mice was used. One or two animals were given the basal ration; for example, 93 per cent. Mellin's Food and 7 per cent. casein; a second group was given this plus 1 c.c. fresh milk to the day's ration; a third group 3 c.c., a fourth 5 c.c., and so on. The gross composition (in protein, salts and total energy value) of these diets did not vary outside the limits found to give normal growth in mice.

The mice receiving no milk grew only very slowly (see the solid line in Charts 1 and 2), although their food was practically identical in gross composition with a food on which normal growth and reproduction had occurred.[6] The addition of 1 c.c. of milk to the day's ration of each mouse increased the growth rate decidedly (Charts 1 and 2 in this paper). The addition of milk made a food adequate to support entirely normal growth in mice, as is indicated in Chart 3. Later, a mixture of 2 c.c. milk and the same food (Mellin's, 93 per cent.; casein, 7 per cent.) was fed to two mice. One mouse grew at a normal rate (Chart 5); the other was evidently a sick animal, since a change to a general mixed diet did not materially affect the rate of gain in weight.

The effect of small quantities of milk on growth was still more clearly indicated in the reaction of mouse 194, which was fed a Mellin-casein-olive-oil mixture (Formula 7, Table 1, this paper) to see whether increasing the total energy value of the ration would hasten growth. The animal remained at nearly constant weight for seventeen days. Then 1 c.c. of milk was daily mixed with the food and the animal grew very rapidly, reaching normal weight for its age in ten days (Chart 6). On the withdrawal of milk from its dietary, all growth ceased at once, to be resumed almost immediately when 1 c.c. milk was again fed.[7]

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6. Column 2, Food B, Table 172, previous paper.

7. Cf. Hopkins, F. G.: Jour. Physiol., 1912, xliv, 425. Hopkins, working with rats, found that milk to the extent of 4 per cent. of the total solids of the food must be added to mixtures of isolated food substances (casein as the protein) in order to secure normal growth. In the present experiments, milk provided 5.8 per cent. or more of the total solids of growth foods.

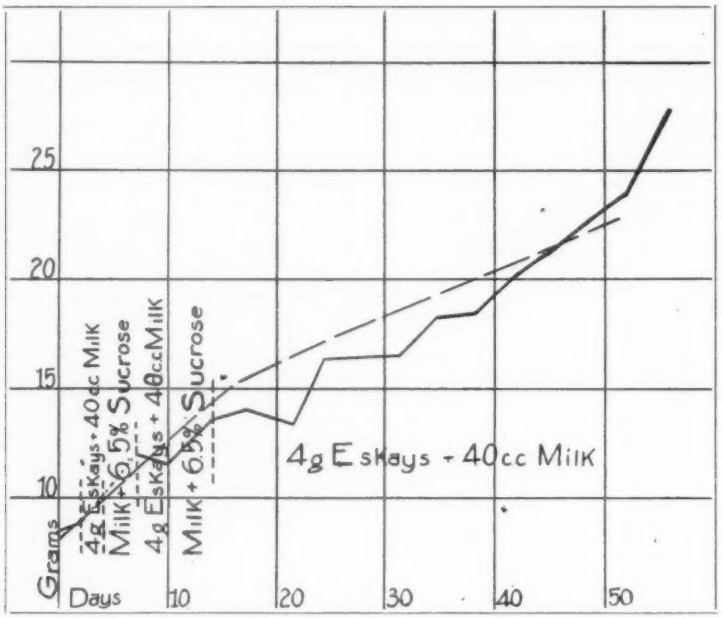

Chart 13.—Mouse 236 ♀, showing growth on a diet of milk plus 6.5 per cent. sucrose and cessation of growth on a diet of Eskay's Food plus 40 c.c. of milk to the usual daily ration (4 gm.). At the end of the first period on the Eskay's-milk mixture, the mouse was so feeble that its diet was changed without preliminary weighing of the animal. It is on this account that the graph is dotted in this and the following period. In the final period this same diet sustained normal growth.

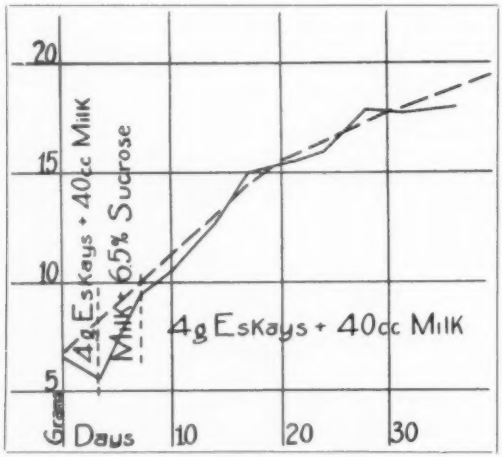

Chart 14.—Mouse 254 ♂, showing normal growth on a mixture of 4 gm. of Eskay's Food and 40 c.c. of milk after an initial loss on this same diet. The interrupted line indicates normal growth for mice.

enrich its protein and salts content. The modifications will be found in Table 2, this paper. It will be noticed that the final food mixtures, after the addition of milk in the proportions indicated in the formulae, are closely similar in gross composition. Slowness of growth cannot be attributed to the presence in food mixtures of casein or of protein-free milk, as both these have been used scores of times in food mixtures which sustained normal growth and reproduction. Since both the gross composition and the substances added have alike proved to be altogether satisfactory in growth foods, failure to grow in these trials must be due to some inadequacy in the nutrients present in the Eskay's Food, or to the absence from it of accessory substances, or to both these factors.

Unlike Mellin's Food, Eskay's must be boiled, as preliminary experiments showed. To every 10 gm. of the food powder, modified or unmodified as the case might be, a few cubic centimeters of distilled water was added to make a thin, smooth paste, the rest of 50 c.c. of distilled water added with constant stirring; the whole heated to boiling, still with constant stirring, and finally heated over boiling water, in a double boiler, for forty minutes. After rapid cooling in a stream of running water and replacing the evaporated water, the milk was thoroughly stirred in.

The previous experiments showed that Eskay's Food, modified to the proper gross composition and fed in a paste with distilled water, was insufficient to induce growth in young mice, or even, except in a single instance, to sustain life for any length of time. Boiling the food did not alter these results. With 3, 5 or 10 c.c. of milk added to the day's ration (4 gm.) the result was the same; very young animals lost weight rapidly, the first group of ten becoming so feeble by the second morning — forty-eight hours' feeding — that their diet was changed without even weighing them. On fresh cow's milk plus 6.5 per cent. sucrose,[8] they rapidly recovered, weighing at the end of two more days from 30 to 60 per cent. more than at the beginning of the experimental feedings (see Charts 7 to 12, inclusive). On a return to the Eskay's-milk diets for the three succeeding days, all the animals either lost weight or just held their own; but though in most cases the relative loss of weight was not serious, the animals were all so evidently ill — with very rough coats, bodies hunched up, hind legs almost useless, and stools diarrheal — that all were again given the sucrose-milk diet for four days, during which very great gains in weight were made, the diarrhea disappeared completely, and the whole appearance of the animals became normal. The Eskay's-milk mixtures were then fed

8. This makes the protein 18 per cent. of the total solids of the food and the salt content 3.8 per cent.; i. e., comparable with that of the other foods.

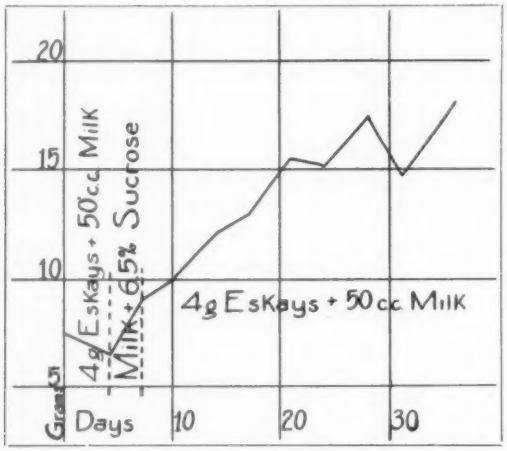

Chart 15.—Mouse 253 ♀, showing normal gain in weight on a mixture of 4 gm. of Eskay's Food and 50 c.c. of milk after an initial loss on this diet, and recovery on a milk-sucrose food.

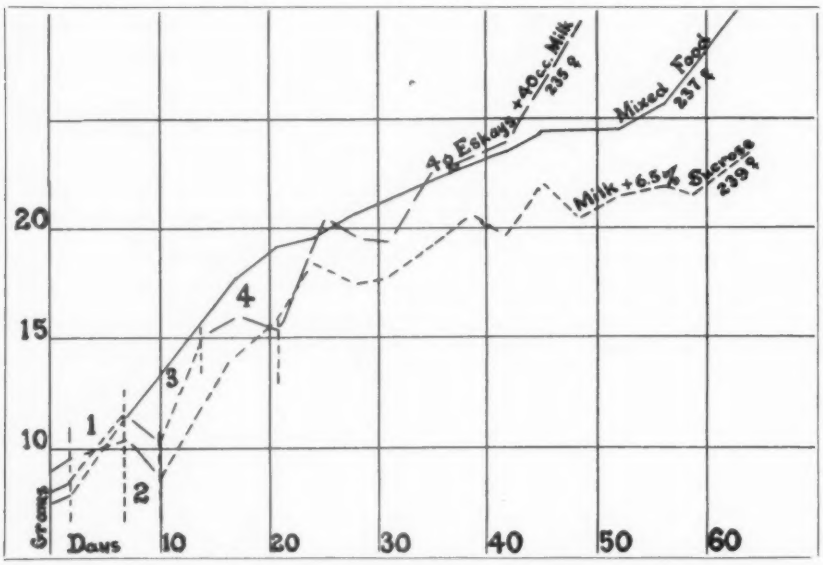

Chart 16.—Comparative growth on three dietaries: mixed food, milk plus 6.5 per cent. sucrose and 4 gm. of Eskay's Food plus 40 c.c. of milk. The last section of the highest line represents a pregnancy curve (Mouse 235 ♀). For diets during the first twenty days see Charts 12 and 17.

once more, this time for a week, with results similar to those in the preceding periods on these diets, but the symptoms of disturbance generally were less marked. The larger animals showed the effects less quickly. Then the whole group of animals was given a mixture of 40 c.c. of milk to 4 gm. of Eskay's,[9] the latter used now without modification, as so large a proportion of milk furnished enough protein and salts to make the resulting mixture of the right composition (see Formula 5, Table 2, this paper). The rate of growth for the succeeding thirty days compared favorably with that of the controls from the same litters on a general mixed diet and with that of two other controls on the milk-sucrose food. During this period there was a tendency to diarrhea on the part of a few of the mice on an Eskay-milk diet, but not continuously or in all the animals. Chart 14 also shows growth on this same mixture and Chart 15, growth for a considerable period on a mixture of 50 c.c. of milk with 4 gm. of Eskay's Food.

REPRODUCTION

Two female mice, 235 and 236, on a ration of Eskay's Food and milk in the proportion of 40 c.c. milk to 4 gm. Eskay's Food, became pregnant, bore and reared the young. Mouse 235 had had the diet twenty-five days and mouse 236 forty-three days before the birth of the young. The litters were numerically small in both cases, two mice in one litter and four in the other, as compared with from eight to twelve in litters of animals on a general mixed diet; but nevertheless, the successful rearing of the young to normal size at the end of the nursing period, is significant evidence that the food mixture in question contained the substances necessary for growth in mice.

CONCLUSIONS FROM FEEDING TRIALS WITH ESKAY'S ALBUMINIZED FOOD

Eskay's Albuminized Food, used strictly as a milk modifier, allows normal growth in mice. In the most satisfactory mixture studied, Eskay's Food furnished 6.1 per cent. of the ration as fed, or 33 per cent. of the actual nutrients (total solids). Milk must furnish more than half the total nutrients if there is to be normal growth.

The absence of growth in animals in whose diet Eskay's Food forms 60 per cent. of the total nutrients, indicates that the character of the nutrients is at fault — that the difficulty is not due simply to the lack of essential accessory food substances. That these last-named essential substances are lacking, however, seemed indicated in the earlier series of feeding trials in which mice showed only minimal

9. This formula closely resembles that recommended by the manufacturers for a child six months old.

growth on a mixture of Eskay's Food with casein and milk salts ("protein-free milk") in which over 95 per cent. of the protein and the same proportion (95 per cent.) of the salts were purified milk products.

In Chart 16 an attempt has been made to summarize the results of these experiments. The cessation of the growth or actual decline on foods in which Eskay's Food furnished 60 per cent. or more of the nutrients is shown in the sections of the graph marked 2 and 4, between vertical dotted lines. The rapid gain in weight when the diet was changed to milk containing 6.5 per cent. sucrose is shown in sections of the graph marked 1 and 3. It will be noted that rapid gain in weight was also made on a ration of 4 gm. of Eskay's in 40 c.c. milk.

In this series also, granulated sugar proved quite as adequate a modifier of cow's milk as the prepared food, as far as the rate of growth and the apparent health of the animals is concerned.

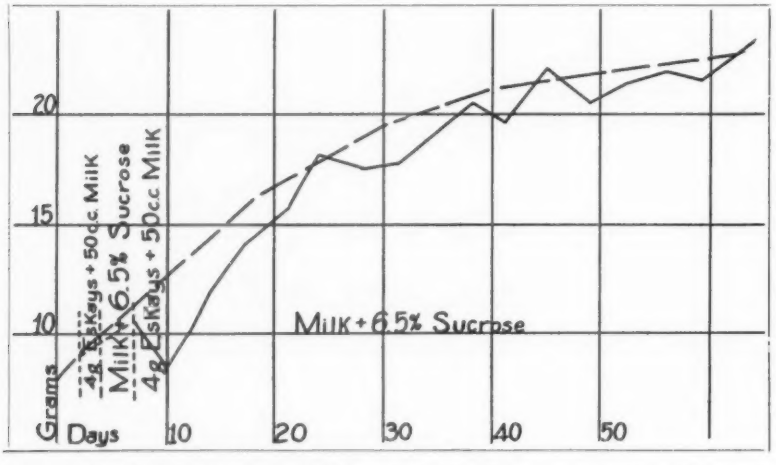

Chart 17.—Mouse 239 ♀, showing normal growth on milk plus 6.5 per cent. sucrose.

SUMMARY

Mixtures of milk and Eskay's Albuminized Food sustain normal growth in albino mice provided the milk furnishes 94 per cent. of the mixture as fed (66 per cent. of solid nutrients).

The lack of growth on Eskay's Food without milk appears to be due to two factors: (1) inadequacy of the proteins to satisfy the nutritive requirements for growth in mice, and (2) absence from this preparation of some accessory substance or substances essential to growth.

Eskay's Food in milk mixtures, as when fed without milk, produces in mice soft stools and sometimes diarrhea.

Mixtures of Mellin's Food and milk also sustain normal growth in mice. Here, too, the milk serves two purposes: to supplement inadequate proteins and to supply essential accessory food substances.

A mixture of 93 per cent. Mellin's Food and 7 per cent. purified casein is adequate for long-continued maintenance, but not for growth; 2 c.c. of milk added to the daily ration (4 gm.) of this mixture, makes a food which sustains entirely normal growth in mice. Here, milk furnishes 5.8 per cent. (including casein, 7.2 per cent.) of the total solids of the diet.

The feces of animals on a diet of Mellin's Food mixtures were always comparatively hard and dry.

A mixture of fresh milk and 6.5 per cent. sucrose also sustained entirely normal growth in young mice. From an economic standpoint at least, this fact deserves consideration.

STUDIES ON INFANT METABOLISM AND NUTRITION

UNDER THE DIRECTION OF L. EMMETT HOLT, M.D., AND DONALD D. VAN SLYKE, PH.D.

The Excretion by Infants of Magnesium Sulphate Injected Subcutaneously *

ANGELIA M. COURTNEY
AND
HELEN L. FALES
NEW YORK

Since Meltzer and Auer demonstrated the narcotic action of magnesium salts injected into the circulation, several clinical applications of this action have been found valuable.

For example, magnesium sulphate has been used intraspinally with excellent results to combat the convulsions of tetanus,[1] in many parts of the world.[2] Magnesium sulphate also induces an anesthesia which permits surgical intervention.[3] It has recently been found that a combination of magnesium sulphate (subcutaneous) and ether (inhalation), produces a surgical anesthesia which is not obtained when either in the same amount is used alone.[4] It may also be mentioned that saturated solutions of magnesium sulphate are employed for the local treatment of burns and erysipelas.

Recently, Behrend[5] has utilized magnesium sulphate, injected subcutaneously into infants, to alleviate the convulsions of tetany. The salt proved remarkably effective in immediately stopping convulsions and reducing the condition of hyperirritability. As the effect was temporary, however, the injections had in the most severe cases to be repeated on a number of successive days. Because of this necessity it appears highly desirable to ascertain the rate at which magnesium sulphate is eliminated after subcutaneous injection. For if the doses require more than twenty-four hours for their excretion, daily repetition of the injection would lead to an accumulation of magnesium in

* Submitted for publication Jan. 8, 1915.

* From the Chemical Laboratories of The Rockefeller Institute for Medical Research and The Babies' Hospital, New York.

1. Meltzer, S. J., and Auer, J.: Jour. Exper. Med., 1906, xiii, 692.
2. See for example, Rocher, Theodore (Cor.-Bl. f. schweiz. Aerzte, 1912, lxii, 969); Stadler, H. (Berl. klin. Wchnschr., 1914, li, 15; ibid., 109); Weintraud, W. (ibid., 1717).
3. Haubold, H. A., and Meltzer, S. J.: Jour. Am. Med. Assn., 1906, lxvi, 647.
4. Meltzer, S. J., and Auer, J.: Zentralbl. f. Physiol., 1914, xxvii, 632.
5. Behrend: Monatsch. f. Kinderh., 1913, xii, 269.

EXCRETION BY INFANTS OF MAGNESIUM SULPHATE INJECTED SUBCUTANEOUSLY

Date	MgO Received			MgO Excreted			MgO	Sulphate Injected (Calc. as H_2SO_4)	Sulphate Excreted in Urine	Sulphate Excreted in Urine in excess of Injected Amount
	Food	Injected	Total	Feces	Urine	Total	Balance			
David W.;* Weight, 5,799 gm.; Age, 5 months										
12/1	0.1186	0.2683	0.3869	0.0772	0.1403	0.2175	+0.1694	0.6513	0.8166	0.1653
12/2	0.1156	0.1878	0.3034	0.0772	0.2603	0.3375	—0.0341	0.4560	0.9895	0.5335
12/3	0.1258	.....	0.1258	0.0698	0.0178	0.0876	+0.0382	.....	0.2387	0.2387
12/4	0.1216	.....	0.1216	0.0698	0.0266	0.0964	+0.0252	.....	0.2273	0.2273
12/6	0.1016	0.2683	0.3699	0.0698	0.1616	0.2314	+0.1385	0.6513	0.8165	0.1652
12/7	0.1198	.....	0.1198	0.0698	0.0237	0.0935	+0.0263	.....	0.1760	0.1760
Joe D.; Weight, 3,845 gm.; Age, 5 months										
12/1	0.0798	0.2683	0.3481	0.0658	(lost)	.....	.....	0.6513	0.9993	0.3480
12/2	0.1033	0.1342	0.2375	0.0658	0.0649	0.1307	+0.1068	0.3257	0.5231	0.1974
12/3	0 1028	.....	0.1028	0.0658	0.0224	0.0882	+0.0146	.....	0.1717	0.1717
12/4	0.1033	.....	0.1033	0.0658	0.0065	0.0723	+0.0310	.....	0.2132	0.2132
12/5	0.1033	.....	0.1033	0.0707	0.0134	0.0841	+0.0192	.....	0.2206	0.2206
12/8-11 (per day)	0.1110	.....	0.1110	0.0840						
Peter L.; Weight, 9,568 gm.; Age, 10 months										
2/2	0.1940	0.1073	0.3013	0.1165	0.0698	0.1863	+0.1150	0.2605	0.7006	0.4401
2/3	0.1925	0.2146	0.4071	0.1165	0.2093	0.3258	+0.0813	0.5211	1.1455	0.6244
2/4	0.1940	.....	0.1940	0.1165	0.0690	0.1855	+0.0085	.....	0.4754	0.4754
2/5	0.1940	.....	0.1940	0.1608	0.0464	0.2072	—0.0132	.....	0.3424	0.3424
2/6	0.1940	.....	0.1940	0.1608	0.0455	0.2063	—0.0123	.....	0.5067	0.5067
Elvira P.; Weight, 3,720 gm.; Age 3½ months										
10/ 9-10	0.100	.....	0.100	....	....	0.0767	+0.0233	.....	0.1443	
10/10-11	0.070	.....	0.070	....	....	0.0426	+0.0274	.....	0.1127	
10/11-12, 1st 12 hrs.	.....	.....	.....	....	....	0.0792	.....	.....	0.2427	
10/11-12, 2d 12 hrs.	.....	.....	.....	....	....	0.0408	.....	.....	0.1042	
10/11-12, altogether	0.085	0.100	0.185	....	....	0.1200	+0.0650	0.2451	0.3469	0.1018
10/12-13, 1st 12 hrs.	.....	.....	.....	....	....	0.1330	.....	.....	0.4897	
10/12-13, 2d 12 hrs.	.....	.....	.....	....	....	0.0814	.....	.....	0.1371	
10/12-13, altogether	0.080	0.200	0.280	....	....	0.2144	+0.0656	0.4902	0.6268	0.1366
10/13-14	0.100	.....	0.100	....	....	0.1126	—0.0126	.....	0.0934	

* In this patient the injections of Dec. 1 and 2 were made 9 hours after the beginning of the 24-hour metabolism periods.

the body. Such an accumulation, in view of the toxicity of magnesium salts, could not be regarded without apprehension.

We have recently had occasion to use magnesium sulphate, in the manner described by Behrend, in treating a number of cases of tetany. The clinical effects were identical with those described by him. The irritability was immediately reduced, and did not return for the greater part of twenty-four hours. Some infants with especially severe convulsions were greatly benefited by the injections. In three cases we have followed the magnesium and sulphuric acid metabolism on the days succeeding the injections. The data are not so full as could be desired, as regards either the number of cases or the number of successive injections received by the individual patients. They are published, however, as they are the only observations available on the question, and the opportunities for obtaining more data are, from the nature of the problem, somewhat uncertain.

The magnesium sulphate was in each case injected subcutaneously with a syringe into the cellular tissue of the abdomen. The concentration of the solutions was 8 gm. of anhydrous $MgSO_4$ (equivalent to 16.4 gm. of Epsom salts) per 100 c.c., the volumes injected varying from 4 to 10 c.c. The children were placed in a comfortable metabolism bed which permitted the collection of urine and feces separately, and the excreta during the succeeding days were analyzed for magnesium and sulphates. The magnesium content of the food was also determined, so that the magnesium balance could be ascertained. The presence of organic sulphur in the food proteins makes a determination of sulphate balance impossible. We can, however, compare the daily excretion of sulphates when no magnesium sulphate is injected, with the excretion in the periods following the injections, and from the increase in these periods decide whether the injected SO_4 is excreted, and the rapidity with which the excretion occurs.

The results indicate the following:

MAGNESIUM

The increase in the elimination of magnesium by the feces is not sufficiently marked to make it certain that an important part of the injected magnesium is eliminated in this way.

In the urine, on the contrary, an increased magnesium excretion immediately followed the injection and practically ceased within twenty-four hours. The relation of the amount excreted to that injected was rather peculiar. A portion of the magnesium given in the first injection was, in two of the three cases, retained. When on the following day, however, another injection was given, an amount equivalent to the second injection was eliminated within twenty-four

hours. In the second case (J. D.) elimination of the second injection was incomplete. In no case was there conclusive evidence that any of the retained magnesium underwent gradual elimination during the succeeding days. The magnesium balance became normal again on the day after the injection. The magnesium is excreted within a period of about twenty-four hours or some of it is permanently retained. In Case 4 a separate collection of the urine and feces was impossible; but the intake and the total excretion are included in the table. In this patient the excretion was measured in twelve-hour periods after the injection. These figures show that the greater part of the elimination occurs in the first twelve hours. The amount excreted was twice as great as in the second twelve-hour period. On the second day, with an injection of 0.2 gm., the retention was no greater than on the first day after an injection of 0.1 gm. During the second twenty-four hours following the last injection the excretion was about 0.04 more than with the same intake before the first injection. On this day, therefore, he disposed of about one-half of the extra amount retained during the days of injection.

In brief, although the data do not cover enough ground to be conclusive, they indicate that the infant organism can incorporate within two successive days as much as one- or two-tenths of a gram of magnesium oxid from the magnesium sulphate injected subcutaneously, and that amounts in excess of this are immediately excreted by the kidneys.

In the doses mentioned in this paper there appears therefore to be no danger of cumulative effect of magnesium if the dose is not repeated more frequently than once in twenty-four hours. The smaller dose referred to is calculated for an average infant of from one to four months and the latter for one from five to ten months. These are equivalent respectively to 0.6 gm. (10 grains) and 1.6 gm. (25 grains) of Epsom salts or one-half the amount of anhydrous magnesium sulphate. Marked physiologic symptoms are usually produced with this dosage and in many cases smaller doses will suffice.

SULPHATE

The injected SO_4 is immediately and completely excreted in the urine. In fact, as will be seen from the last column, the rise in sulphate excretion on the day of injection corresponded to even more than the amount injected in two cases (the second injection of D. W. and the first of J. D.). After the other five injections the rise in excretion approximately equaled the amount injected.

TUBERCULOSIS AS A DISEASE OF THE NEWBORN*

CLIFFORD G. GRULEE, M.D., AND FRANZ HARMS, M.D.
CHICAGO

It seems to us that of all the chapters in medicine the most neglected is that of diseases of the newborn. We are only beginning to get here and there a little light on the clinical pictures here presented, and this shows us that the newborn infant is affected clinically by the same infections in a much different way from the older infant. It is for this reason that we wish to call attention to the subject of tuberculosis in the newborn, with the hope, not of adding a great deal to the literature, but of stimulating interest in a subject which has been so long neglected.

Much has been said and written about tuberculosis in children, and not a little about congenital tuberculosis. This has been a field which has been much studied by pathologists, but in which, as yet, the clinician has taken little interest. This is probably due to two causes: (1) the hopelessness of the case should the diagnosis be made, and (2) the extreme difficulty of making the diagnosis.

Though recognizing these facts, we feel that an attempt to add something to the clinical picture of tuberculosis may eventually be of practical as well as of scientific interest.

E. R. entered Cook County Hospital, obstetrical ward, Aug. 30, 1912, aged 20 years. She was transferred from the obstetrical ward to the woman's venereal ward because of a leukorrhea which, though suspicious, yet contained no organism which could be positively identified as the gonococcus. So far as the history shows, examination was negative except for an old, apparently healed, tuberculosis of the hip. It might be stated here that she left the hospital in good health and was known to be in apparently good health for several months after leaving the hospital, a notable fact in this case.

September 6 she gave birth to a baby, weighing 6 pounds, 8 ounces. This child was of a fair degree of nutrition and apparently at term. The temperature curve is best shown in the accompanying chart (Fig. 1). It will be noted that from the second day on the child ran an irregular temperature, varying between 98.6 F. and 105 F. During the first night the baby was very cross, and continued in that state for several days. The child did not get much from the breast and often refused to take the bottle feeding. Aside from warm packs for temperature and an occasional enema, no treatment was given. At 8:40 a. m. on the 13th the child had its first convulsion. It had two more convulsions before noon of that day. At that time the child was seen by one of us and the general examination revealed a child in a fair state of nutrition, apparently unconscious, with facial twitchings and eyes turned upward, first to the one side and then to the other. There were no râles distinguishable in the lungs; the heart area was not increased, nor were any murmurs detected. The liver was about 2 inches below the costal margin in the right mammary

* Received for publication Jan. 30, 1915.

line. The spleen was palpable. There was no enlargement of the epitrochlear glands and no infection that could be detected of the umbilical vessels. Despite the latter fact, a tentative diagnosis of septic infection was made, the possibility of such a condition complicating congenital syphilis being considered. From this time the child was given albumin milk. It had to be fed with a medicine dropper and regurgitated most of what was given. No further convulsions were recorded until the evening of the same day, when four more convulsions occurred within an hour, and a fifth before midnight. The amount of food taken was very small, at times being only 1 or 2 drams. For the control of the convulsions 2 grains of chloral hydrate rectally were administered on the morning of the 15th. This was repeated again in the afternoon and again in the evening. After the administration of the chloral hydrate the convulsions became much fewer, but did not cease altogether until the evening of

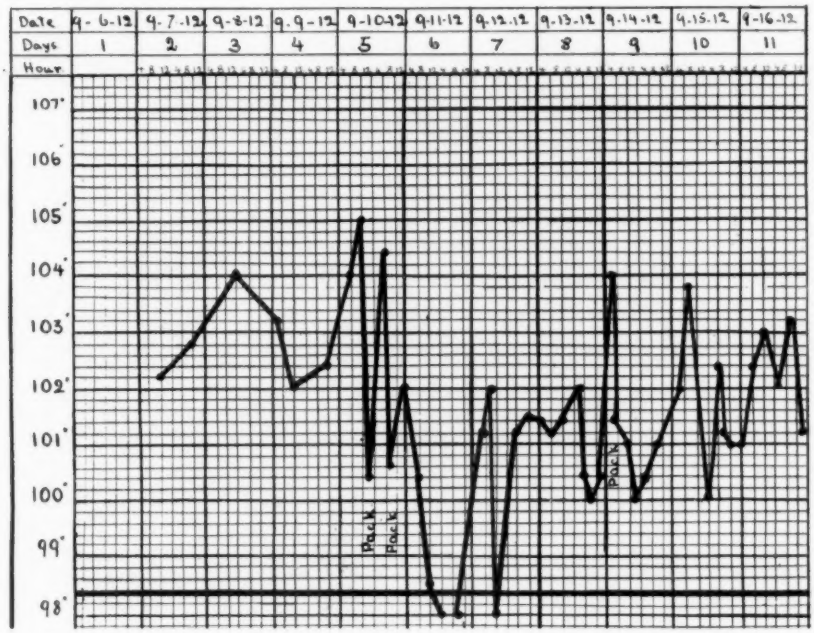

Fig. 1.—Temperature curve in tuberculosis of the newborn.

the 15th, after which time no further convulsions occurred. The child died at 11 p. m. on September 16. At necropsy the following was found:

Baby R., aged 11 days.

Body is that of an emaciated infant, about 50 cm. long, weighing about 7 pounds. Post-mortem lividity and rigor are not well marked. Eyes, ears and nose are negative. No marks of injury to head. Fontanelles large and gaping.

Brain: Congestion of superficial vessels over the right hemisphere. Cerebrospinal fluid increased slightly in amount. Cross sections of pons, cerebrum and medulla are negative. Ventricles are normal in size and contain a normal amount of fluid.

Thorax: Pleural cavities contain a small amount of clear, straw-colored fluid. Pericardium contains a small amount of a similar fluid. Pleura and pericardium are smooth and shiny.

Lungs: Collapse only slightly. Surface is uneven, showing depressions — irregular — and of a bluish color. Everywhere crepitant. Lungs are studded with a few scattered nodules, yellow-white in color, firm, up to size of sago seed. Cut surface: Moist, red and shows similar nodules, peribronchial thickenings. Peribronchial glands markedly enlarged (Fig. 2).

Heart: Normal size. Musculature reddish-brown. In wall of left ventricle is a small white nodule, size of a pinhead. Aortic, pulmonic, tricuspid, and mitral valves are normal.

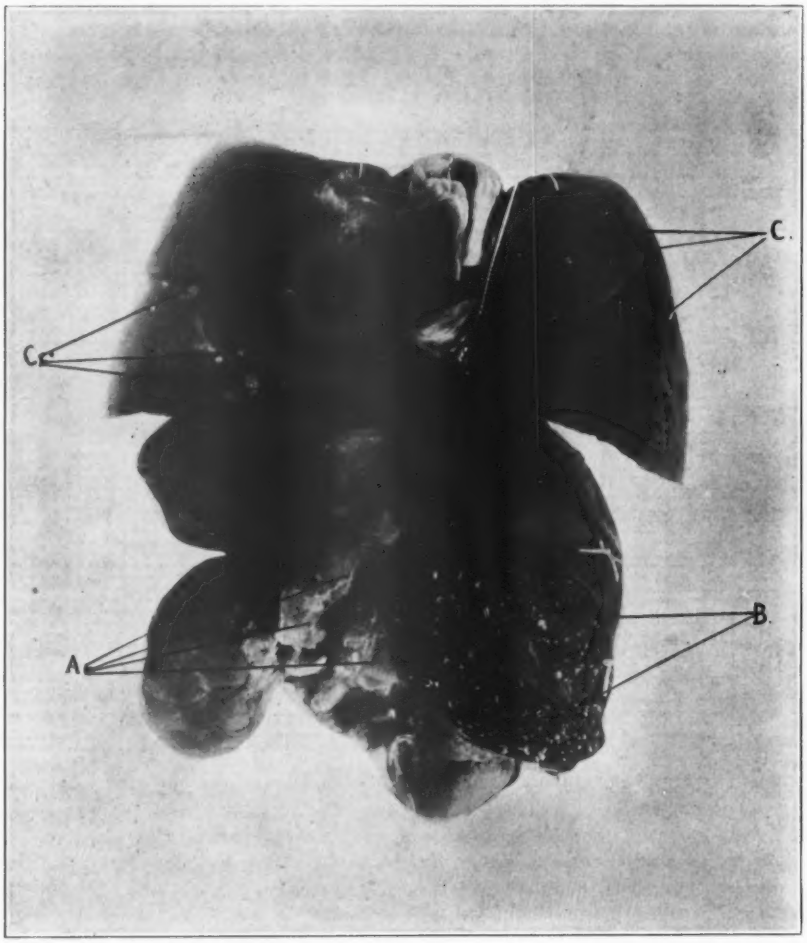

Fig. 2.—Tuberculosis of the newborn. *A*, tuberculous mesenteric and peri-portal glands. *B*, miliary tuberculosis of the spleen. *C*, tubercles in the lungs.

Abdomen: No free fluid. Peritoneum smooth and shiny. Ileocecal region is free. In region of gall-bladder is a massive growth, surrounding the gall passage, the size of a walnut, nodulated with masses up to size of a pea.

Liver: About normal size. Studded with a few nodules, both on the external and on the cut surface, similar to those described. Cut section: Lobules are very indistinct; parenchyma cloudy.

Pancreas: Head is imbedded in the mass surrounding the gall passage and is studded with similar nodules.

Spleen: Firm. Studded thickly with nodules up to size of sago seed (Fig. 2).

Kidneys: Fetal lobulation. Markings fairly distinct. A few nodules similar to those described.

Adrenals: Fairly large and show an occasional nodule.

Stomach: About normal size. Along lesser curvature are nodules up to size of small pea.

Intestines: Negative.

Mesenteric Glands: Enlarged, soft, red-gray. Microscopically, the nodules are seen to be made up of a coalescence of tubercles. A great number of tubercle bacilli are present.

Anatomic Diagnosis: Miliary tuberculosis of all organs, with tuberculosis of mesenteric lymph glands.

EPICRISIS

We have, then, the child born of a mother with supposedly a healed tuberculosis, whose only subjective symptom was a severe leukorrhea of unknown cause. The mother survived the child (an unusual thing in these cases) many months at least, and probably is still living. A baby born apparently at term and apparently healthy, had as early as fifteen hours after birth a high temperature, which continued irregularly until death. There was a distinct tendency to regurgitation, and on the seventh day generalized convulsions developed, which continued up to twenty-four hours before death. Death occurred on the eleventh day. On physical examination the child showed only an enlarged liver and enlarged spleen. At necropsy a generalized tuberculosis affecting most markedly the abdominal organs, and especially the periportal lymph-glands, liver and spleen was found. The tuberculosis was miliary in type, but the stage of the tubercles suggests that it had already begun *in utero*. Many tubercle bacilli were found in all sections of tuberculous areas (Fig. 3).

GENERAL CONSIDERATION

Tuberculosis of the placenta has been described by many authors. This is of importance in relation to tuberculosis of the fetus in proportion as the fetal or maternal portion of the placenta is involved. It is certain that in many cases only the maternal portion is involved, the fetal remaining uninfected by the tuberculous condition.

As to the relative frequency of tuberculosis of these two portions, it has been impossible for us to judge from the authorities consulted. It is worthy of note, however, that both placental tuberculosis and congenital tuberculosis are being reported with increasing frequency within the last few years.

The most exhaustive work on human congenital tuberculosis is that of Warthin and Cowie[1] published in 1904. They separate the authentic cases from those not sufficiently supported by evidence and find only five cases of certain congenital tuberculosis.

1. Warthin and Cowie: Jour. Infect. Dis., 1904, i, 140.

Cases of unquestioned tuberculous bacillosis have been reported by Bugge,[2] Londe[3] (Case 5), Thiercelin and Londe,[4] and Schmorl and Kockel.[5] Nothing further need be said of this condition here except that it may form the basis for the development of cases of congenital tuberculosis which die after the first few weeks, if in fact such exist, about which there is a reasonable doubt.

Of the cases of tuberculosis of the newborn judged from a pathologic standpoint there are two forms, both of which of course are miliary in type: (1) pulmonary and (2) abdominal. (It should be stated here that all cases of this age reported as localized tuberculosis lack evidence necessary to be convincing.)

Fig. 3.—Showing tubercle bacilli in the liver tissue.

The first form is represented by the cases of Harbitz[6] and Brindeau.[7] In each of these the thoracic organs alone were affected and there was no involvement below the diaphragm. In the case of Harbitz the mother had an endometrial tuberculosis: in that of Brindeau no mention is made of the location of the tuberculosis in the mother. Brindeau's patient died on the twelfth day and showed large caseous nodules in the base of the lungs; Harbitz' patient died on the twenty-fifth day and showed miliary tuberculosis of the lungs with caseous peritracheal glands. There can be little doubt that the case of Brindeau is one of congenital tuberculosis. A doubt, however, may be raised as to that of Harbitz, because of the age to which the child lived, but

2. Bugge: Beitr. z. path. Anat. u. z. allg. Path., 1896, xix, 433.
3. Londe: Rev. de la Tuberc., 1893, i, 125.
4. Thiercelin and Londe: Centralbl. f. Chir., 1894, xxi, 563.
5. Schmorl and Kockel: Beit. z. path. Anat. u. z. allg. Path., 1894, xvi, 313.
6. Harbitz: München. med. Wchnschr., 1913, lx, 741.
7. Brindeau: Bull. Soc. d'Obst. de Paris, 1899, ii, 235.

considering the presence of the endometrial tuberculosis it is altogether likely that the infection was congenital. In these cases one must look for a port of entry for the infection other than the umbilical vessels. In other words, placental infection need not be regarded as essential to this form. Much more likely would seem to be an aspiration of infected liquor amnii or vaginal secretions at the time of birth. While this might account for the location of the infection in the infant it would not coincide with the advanced nature of the lesion such as is seen in the case of Brindeau or perhaps in that of Harbitz. From the stage of the lesions it is altogether likely that the infection occurred *in utero*. If this be true, then the only plausible explanation is that it is due to an infection of the liquor amnii, an infection which might readily take place with an endometrial tuberculosis as in the case of Harbitz. There are two objections to this theory; the first is that the fetus is not supposed to respire *in utero* and hence the infected liquor amnii would not come in contact with the alveoli so as to produce lesions in the lungs. To explain the case of Harbitz it is only necessary to recall the recent work of Ghon,[8] Wollstein and Bartlett,[9] and others, which goes to show that the primary lesion may be in the bronchi (which are exposed to the liquor amnii) from which the neighboring lymph glands about the hilum are infected, and from these the lesion extends to the rest of the lung. Such an explanation, however, will not hold for the case of Brindeau. If we accept the recent work of Addison and How[10] and of Ridella[11] it is altogether likely that the infant does respire *in utero,* in which case the infection of the deeper portions of the lungs would be readily explained. The other objection is that in both these cases the intestines have remained free. It is a well-known fact that the intestinal mucosa in extra-uterine life is quite resistant to tuberculous infection and it is quite likely that such is the case *in utero,* especially when we take into consideration that in its parasitic state the fetus does not use the intestinal canal for the purpose of supplying nutriment to the body.

The second or abdominal form might be termed the general miliary form. While the lesions are more marked in the abdominal cavity the thoracic viscera are always involved. We have been able to find in the literature ten cases of this form of tuberculosis[12] of the newborn other

8. Ghon: Der primäre Lungenherd bei der Tuberkulose der Kinder, Berlin and Vienna, 1912.

9. Wollstein and Bartlett: A Study of Tuberculous Lesions in Infants and Young Children, Based on Post-Mortem Examinations, Am. Jour. Dis. Child., November, 1914, 362.

10. Addison and How: Am. Jour. of Anat., 1913, xv, 199.

11. Ridella: Ref., Ztschr. f. Kinderh., 1914 (Ref.), viii, 342.

12. The case of Strauss (Ztschr. f. Fleisch- u. Milch-Hygiene, 1910, xx, 129) quoted by V. Reuss (Die Krankheiten des Neugeborenen, Berlin, 1914, p. 452) proved on investigation not to be one of human tuberculosis.

than the one here reported (Sabouraud,[13] Honl,[14] Lehmann,[15] Ustinow,[16] Auché and Chambrelent,[17] Stoeckel,[18] Wollstein,[19] Lobenstine,[20] Rollet,[21] and Möller[22]). Of these, only Wollstein's patient died of a disease other than the tuberculosis. In this instance the cause of death was bronchopneumonia and at necropsy scattered tubercles were to be found. This case suggests a close relation with the bacillosis previously mentioned. In all of this group the most advanced lesions were to be found in the periportal glands and the involvement of the abdominal organs was greater than that of the thoracic. In many instances the placenta was found to be involved. We have here unquestionably infection through the umbilical vessels.

In all the cases of both these groups tubercle bacilli were found either in the tissues or by animal experiment.

The clinical data are very scant. The cases of Stoeckel,[18] Sabouraud,[13] Lehmann,[15] Honl,[14] Ustinow[16] Auché and Chambrelent[17] and Wollstein[19] consist only in pathologic reports, the day of death being given in order, fourteenth, eleventh, first, fifteenth, uncertain, twenty-sixth and nineteenth. Of the other cases, two simply give besides the pathologic findings, the weight at birth and the day of death. In the case of Harbitz[6] the child weighed 1,400 gm., was 47 cm. long, and died on the twenty-fifth day. In that of Rollett[21] the child weighed 3,600 gm., was 51 cm. long, and died in forty-eight hours. The other three cases are somewhat more specific in their statements. That of Brindeau[7] occurred in a baby whose birth weight was 2,150 gm. It was taken from the mother immediately. The child was cyanotic, somewhat edematous, and had a rectal temperature of 36 C. It remained cyanotic and died on the twelfth day. The spleen was normal in size, the liver somewhat enlarged and hard, and at necropsy was shown to weigh 190 gm. Miliary tuberculosis of the lung was found. The sections were very rich in bacilli. The second case is that of Lobenstine.[20] It occurred in a bottle-fed baby who died on the nineteenth day. At birth the child weighed 2,100 gm., at time of death, 1,650, showing a loss of 450 gm. The child was bottle-fed, showed a subnormal temperature throughout life, and had no cough nor diarrhea. An acute miliary tuberculosis was found at necropsy. The third case is that of Möller.[22]

13. Sabouraud: Compt. Rend. Soc. de Biol. de Paris, 1891, xliii, 674.
14. Honl: Cited by Warthin and Cowie (Note 1).
15. Lehmann: Berl. klin. Wchnschr., 1894, xxxi, 646.
16. Ustinow: Arch. f. Kinderheilk, 1898, xxv, 66.
17. Auché and Chambrelent: München. med. Wchnschr., 1898, xlv, 616.
18. Stoeckel: München. med. Wchnschr., 1904, li, 177 and 454.
19. Wollstein: Congenital Tuberculosis: Arch. of Ped., 1905, xxii, 321.
20. Lobenstine: Bull. Lying-In Hosp., City of N. Y., 1905, ii, 9.
21. Rollett: Wien. klin. Wchnschr., 1913, xxvi, 1274.
22. Möller: Arch. mens. d'Obst. et de Gyn., 1914, iii, 1.

The temperature was normal throughout. Respiration was short and labored, but there were no cyanotic attacks. The infant did not nurse well. The mother left the hospital with a tuberculosis of the uterus and died within two months of a general miliary tuberculosis.

From these meager data it is absolutely impossible to draw any definite conclusions as to the symptomatology of tuberculosis in the newborn. It is evident that Lobenstine's patient was a premature baby, or at least one congenitally debilitated, which would account very well for the subnormal temperature which existed throughout, since such an occurrence is not at all rare in infections of infants of this type. One would scarcely expect such a reaction in a baby of the size and vigor of the one we report. It is not at all impossible, too, that the early temperature in our case had nothing whatever to do with the tuberculosis, but was simply the so-called "inanition fever," which is so frequently encountered at this time. The enlargement of the liver, as mentioned by Brindeau, and present in our case, is a suggestive symptom, especially when combined with an irregular temperature existing from birth on. The combination of enlargement of the spleen, high, irregular temperature and enlargement of the liver, together with tuberculosis in the mother, is suggestive. In all cases except the one here reported and that of Möller the tuberculosis in the mother was as severe as that of the child, the date of death of the parent being only a few days before or after that of the child.

DIAGNOSIS

In the diagnosis of this affection very little can be expected from the ordinary tests. In the newborn the Pirquet test has proven negative almost without exception. Zarfl reports a positive *Stich-Reaction* in a baby, 17 days of age. Recently at the County Hospital one of us has been able to obtain a positive von Pirquet reaction in a premature infant, 16 days of age, whose mother was in the tuberculosis hospital. In another instance the reaction was positive on the nineteenth day. This, however, would give us little encouragement in the first few days of life, and hence the tuberculin reaction must probably be disregarded as a definite diagnostic means at this time. Since in many instances tubercle bacilli are found in the blood, a blood culture or the injection of blood into animals might be suggested. This offers three difficulties: first, that of obtaining blood from these young infants; second, the fact that the presence of the condition is rarely suspected, and, third, any animal thus experimented on succumbs to the tuberculosis only after the patient. It would seem, then, to be very difficult to arrive at a certain diagnosis in such cases, even though the case were suspected.

PROGNOSIS AND TREATMENT

As to the prognosis, tuberculosis of the newborn has been up to this time always fatal so far as we can tell. Sitzenfrey[23] reports four cases of tuberculosis of the placenta, however, in three of which the infants died at six weeks, three and six months, respectively; but the fourth remained alive during nine months' observation, and showed no symptoms of tuberculosis. It would seem, then, that the mere presence of tubercle bacilli in the placenta did not mean a tuberculosis of the fetus in all instances. The duration of life has varied from twenty-four hours to twenty-five days. It is very likely that children suffering with congenital tuberculosis may live as long as three to six months. The only clinical data on any case approaching this age is that given by Veszpremi.[24] The premature infant died on the thirty-seventh day, and from birth on had a temperature varying between 36.5 to 38.3 C. (from 97.7 F. to 101 F.).

Of course, treatment at present, in a condition of such extreme severity and of the nature of which tuberculosis of the newborn is, must necessarily be without results.

122 South Michigan Avenue—4800 North Western Avenue.

23. Sitzenfrey: Die Lehre von der kongenitalen Tuberkulose mit besonderer Berücksichtigung der Plazentartuberkulose, Berlin, 1909.
24. Veszpremi: Centralbl. f. allg. Path. u. path. Anat., 1904, xv, 483.

PROGRESS IN PEDIATRICS

LITERATURE OF 1913 AND 1914 OF INFECTIONS OF THE GENITO-URINARY TRACT IN CHILDREN

RICHARD M. SMITH, M.D.
BOSTON

VULVOVAGINITIS

Sources of Infection

Taussig,[1] in a report of sixty-six cases of vulvovaginitis treated in the Washington University Gynecological Dispensary, writes that "a correct appreciation of the etiological factors can only be obtained by considering the anatomy and histology of the lower genital tract in girls before puberty." The epithelium is not only much more delicate than in the adult, but is also far more open to infection, owing to the small size of the labia majora and the absence of hair. "In analyzing the possible causes of this infection we must consider first the source, and second, the manner of transmission." "The first question is whether the parents are the source of infection." In only two cases did the mother have a discharge, and further, only two patients were under 1 year of age, at which time they are most exposed to infection by the mother. He considers that "other already infected girls of the same age" are "the most important factor in the spread of the disease."

Under manner of transmission he considers rape, which is rare; the hand of an infected mother or other person, which is not common, and finally clothing, which is unlikely because of the inability of the gonococcus to withstand drying. "Being primarily concerned with home cases" he leaves out the common bath-tub as a means of spreading the disease, but does lay great stress on the danger of infection from the ordinary water closet seats which are built "so high that the smaller children in using them are forced to have their genitals and clothing rub over a considerable portion of the seat." The greatest danger is in the tenement houses and public schools. Taussig does not give "figures regarding etiology from the social service investigation of the cases, because the evidence on which they were based varied so much in reliability." However, "forty-seven out of sixty-six girls were of school age or over."

1. Taussig, F. J.: Am. Jour. Med. Sc., 1914, cxlviii, 480.

R. M. Smith[2] has studied the sources of infection through home visits by a social worker, and gives the following table based on thirty cases:

Undetermined	5 cases	= 16.6 per cent.
Hospital infection	1 case	= 3.3 per cent.
Playmates	4 cases	= 13.3 per cent.
Assault	4 cases	= 13.3 per cent.
Other members of family infected	16 cases	= 53.3 per cent.

Over half the cases were apparently due to infection from other members of the family, in which Smith's findings disagree with Taussig's opinion, though the two agree that hospital and institutional cases are a minority. Smith says, however:

> Bad habits are taught young children by those older than themselves, in some instances by those old enough to understand fully the significance of sexual matters. Masturbation is extremely common among the infected cases. Whether as a result of this or not . . . many of these children are sexually precocious. . . . I am confident that as we learn more intimately the history of infected children the less often we shall have to attribute the infection to toilets or other indefinite sources.

The same author also emphasizes the great influence of feeble-mindedness both in the children and in adults, finding that "in five of our [thirty] patients the infection is directly traceable to feeble-minded individuals, and two other patients were themselves feeble-minded."

Barnett[3] has analyzed fifty cases from the Mount Sinai Hospital in New York, and gives the following figures:

Discharged from hospital or other institution	36 per cent.
Sleeping with mother who has a vaginal discharge	18 per cent.
Sleeping with sister who has a vaginal discharge	10 per cent.
Cases of unknown origin	36 per cent.

Spaulding,[4] reporting eighty-three cases from the special clinic at the Children's Hospital in Boston, gives a somewhat similar list:

Discharged from hospital	21 per cent.
History of discharge in parent	35 per cent.
History of contact	4 per cent.
Not discovered	40 per cent.

Sinclair[5] examined eighty-three babies at the Babies Hospital of Philadelphia and found that 19 per cent. were infected or highly suspicious on admission, and that 8.4 per cent. developed the disease in the hospital or the convalescent home.

On the whole, the recent trend, notwithstanding the great importance of hospital and institutional epidemics, would seem to place the

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2. Smith, R. M: Vulvovaginitis in Children, Am. Jour. Dis. Child., November, 1913, p. 355.

3. Barnett, N.: Arch. Pediat., 1913, xxx, 650.

4. Spaulding, Edith Rogers: Vulvovaginitis in Children, Am. Jour. Dis. Child., March, 1913, p. 248.

5. Sinclair, J. F.: Arch. Pediat., 1914, xxxi, 29.

ultimate responsibility further back, in the home, the school and the street or playground.

We must recognize that we are dealing not only with a medical disease but also with a social evil, and that the medical treatment alone is only a part of what needs to be done. This means that we must establish a close cooperation with the social workers in their attempt to solve the delicate questions of venereal disease.[5]

Prophylaxis

The medical profession is just beginning to realize the great number of endemic cases in the cities and the difficult nature of the problem. The health commissioner of New York, Dr. Goldwater, has recently made a preliminary report on the question, of which the following is an extract.[6]

That a form of vaginitis which is bacteriologically indistinguishable from gonorrheal vaginitis is a common condition among children in this city and elsewhere, is well known to clinicians.

If a serious attempt were made to exclude from school all children who suffer from vaginitis, it would be necessary (*a*) to establish the machinery by which a complete physical examination of all female school children could be made; (*b*) to convince the public of the necessity of the proposed measure; (*c*) to establish the legal and moral right of the city authorities to require such an examination. Furthermore, inasmuch as the condition in question is one which, notwithstanding the most intensive treatment, often persists for months and even for years, it would be necessary to inaugurate a method by which the education of excluded children could be continued. The only logical method would be to send private teachers into the houses of infected children.

The questions here involved are among the most difficult and puzzling with which public health administrators have to deal. I am not aware that the problem has been solved anywhere, and can only promise, on behalf of this department, unremitting attention to it, in the hope that a working program may ultimately be formulated.

Taussig[1] writes that "Gonorrhea in little girls . . . is simply the result of inadequate sanitary precautions," and offers the following suggestions "as additional preventive measures for the control of this disease":

1. The instillation of a drop of 2 per cent. silver nitrate solution in the vestibulum vaginae of all new born girls whose mothers show evidence of gonorrhea.
2. Making vaginitis in children a disease reportable to the Board of Health. . . . We would not be justified in excluding the children from school for eight or nine months . . ., unless there existed a special school . . . where [they] could be given . . . instruction.
3. Instruction of parents of infected children through the visiting nurse regarding preventive measures to limit the infection.
4. Investigation by the visiting nurse as to the probable origin of the infection in each case with a view to excluding this factor from contaminating other children in the same house.
5. The adoption of a U-shaped seat with low bowl and other precautionary measures to prevent the spread of infection through the public lavatories in schools, playgrounds, comfort stations and tenements. I consider this last named suggestion the most important of all. . . .

6. Goldwater, S. S.: Modern Hospital, 1914, ii, 305.

Taussig also as well as Barnett[3] advises the use of paper coverings for lavatory seats.

These are the most radical recommendations yet made. Most authors[4, 2] content themselves with keeping the children out of school for a week or two, and warning the teachers and school nurses when they go back that they are not to use the toilet. G. G. Smith[7] adds that with "this danger removed [i. e., the common toilet] they are less likely to be a focus of infection while in school than when playing untended on the street."

Diagnosis

Sinclair[5] examined eighty-three girls under 3 years of age consecutively admitted to the Babies Hospital of Philadelphia, and reports that scraping the vagina with a platinum loop was the most efficient method of obtaining a positive smear. Rinsing of the vaginal vault with a 1 : 5,000 mercuric chlorid solution and examination of the centrifugalized sediment of the collected fluid was not satisfactory. Of the positive or suspicious cases 21.7 per cent. would have escaped detection without examination with the urethroscope.

Barnett[3] uses a speculum and mirror to aid him in getting scrapings of the vagina with a platinum loop.

Treatment

The general opinion seems to be that children with vaginitis do better if they are kept in bed or very quiet during the first few weeks and then if particular attention is paid to improving their general condition. Further than this there is the greatest possible disagreement as to treatment. Kerley,[8] as an example of one type of procedure, uses nothing but a cleansing douche of warm boric solution four times a day followed by a thorough drying and dusting with boric acid starch and zinc. He believes "that gonorrheal vaginitis is a self-limited disease, and that something takes place during the child's life that makes the vagina less hospitable to the gonococcus." Hamilton[9] also, as far as the local treatment goes, does nothing but "keep the patient clean and dry."

Barnett[3] uses a daily permanganate douche given at home by the mother or a visiting nurse, and follows it three times a week by an endoscopic treatment of the cervix and vagina with Lugel's solution. He also gives hexamethylenamin. He concludes that

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7. Smith, George Gilbert: The Treatment of Gonococcus Vulvovaginitis, with Further Observations on the Value of the Complement Fixation Test in the Management of this Disease, Am. Jour. Dis. Child., March, 1914, p. 230.
8. Kerley, C. G.: Arch. Pediat., 1913, xxx, 702.
9. Hamilton, A.: Arch. Pediat., 1913, xxx, 702.

The first important fact to bear in mind in treating this disease is the infection of the cervix and the difficulty of reaching this nidus of infection by the ordinary methods of treatment. Any infection of the cervix is difficult to treat because of its inaccessibility to the child. The simplest method . . ., irrigation of the vagina, is neither practical nor of great value. The affected parts are not reached directly, and the treatment must be continued for too long a time . . . Direct applications must be made to the deep point of infection. Cures can be obtained by the treatment of the cervix . . . in a short period of time, and strong solutions can be applied to the cervix and vaginal walls directly without causing any pain.

Spaulding,[4] G. G. Smith,[7] and R. M. Smith[2] all favor a daily cleansing douche followed by the instillation of some silver preparation. G. G. Smith[7] adds that after the injection the child should lie on her back for at least fifteen minutes with her hips elevated and thighs pressed together. He also, in persistent cases, makes direct applications to the cervix through an endoscope, using either tincture of iodin or a silver solution. Taussig[1] omits the douche, but uses locally 25 per cent. argyrol or a solution of silver nitrate, from 2 to 4 per cent.

Rubin and Leopold[10] are the strongest exponents of the necessity of endoscopic treatment. They have made a careful study of fifty cases "under extended observation and under various treatments, including . . . all known therapeutic remedies." They made use in every case of the electric lighted female urethroscope and "have shown that the infection involves the vagina and the vaginal portion of the cervix." They suggest that:

1. It is important to determine the extent of the deep lesion before any active treatment is begun. For this purpose the electric lighted female urethroscope should be used.
2. By means of the same instrument appropriate medication can be carried out.
3. When irrigations are resorted to the douche tip or catheter should enter the vagina at least 1½ inches.
4. Applications by means of swabs used alone are useless and injurious.
5. When strong silver solutions are applied to the cervix and vagina it is well to keep the patient in bed for a few days.

Mattisohn,[11] however, considers that "cervix involvement is not proven."

There is also considerable disagreement about the use of vaccines. Hamilton[9] says she is "still enthusiastic in the treatment of recent cases by vaccines." Comby and Condat[12] give the case reports of sixteen girls with gonorrheal vulvovaginitis and one boy with urethritis, all treated with Nicolle's vaccine, which is a mixture of one part gonococci to nine parts of a similar symbiotic Gram-positive coccus. "The hos-

10. Rubin, I. C., and Leopold, J. S.: On the Cause of the Persistence of Gonorrheal Vulvovaginitis in Children, Am. Jour. Dis. Child., January, 1913, p. 58.
11. Mattisohn: Arch. f. Dermat. u. Syph., 1913, cxvi, 817.
12. Comby, J., and Condat: Arch. de méd d. inf., 1914, xvii, 419.

pital patients remained in bed," but "all local treatment was omitted." "Improvement is in general rapid. After the second injection, sometimes after the first, the diminution in the discharge is very clear; progress thereafter is slower and less regular, and seven injections at least are necessary to obtain a satisfactory result." In recent cases the authors obtained an apparent cure, "but the results in older girls were uncertain." "This method . . . allows the omission of douches, so badly tolerated . . . and so often impossible to entrust to the mother." "In certain more tenacious forms the two treatments can . . . be combined; but, in recent cases, vaccine therapy would seem sufficient."

Spaulding[4] has tried out vaccines in thirty-five cases, in addition to the local treatment. Autogenous preparations she found no better than stock. She concludes that "in our hands vaccine has not proved a specific . . . However, we do feel that it is an additional factor for good . . ."

Barnett,[3] on the other hand, treated fourteen of his fifty cases "with vaccines at intervals of three to seven days in doses of from 5 to 100 millions," and reports that "none of my cases treated with vaccines showed any improvement as to the vaginal discharge."

Heyman and Moos[13] have reached a similar opinion, saying "in open lesions of urethral and uterine mucous membranes it has failed entirely."

Hamburger[14] reports on twenty-one cases in all of which the patients were kept in bed without local treatment during the administration of vaccines. "Five children were given three to four intravenous injections. To this method . . . we must take exception because of the severe . . . reaction. All other injections were intramuscular . . ." He concludes that "The treatment of vulvovaginitis by vaccines alone is absolutely and entirely insufficient to replace local treatment."

Reinfection or Relapse

Spaulding[4] is frankly pessimistic in her belief as to the great frequency of relapse. Of twenty-six cases "all but two had a recurrence of the discharge after intervals varying from three weeks to six months, and one of these two had a vulvovaginal abscess after the discharge had ceased, strongly suggesting that the infection was still present." "We believe . . . that the disease may extend over many years, during which time there may be many recurrences, and that the periods

13. Heyman, H., and Moos: Monatschr. f. Geburtsch., 1913, xxxvii, 632.
14. Hamburger, R.: Deutsch. med. Wchnschr., 1914, xl, 759.

of latency may be as long as eighteen months," and "that the most efficient treatment does not insure a permanent cure."

Mattisohn[11] says that "the prognosis [is] . . . rather favorable for a final cure, [but that] proper treatment takes at least four to six months, often much longer, a permanent cure can be spoken of only if after months or years no gonococci are recognizable."

Barnett's[3] definition of cure is three negative vaginal smears one week apart.

G. G. Smith,[15] in his first paper, writes:

> A study of twenty-five cases of vulvovaginitis by means of the complement fixation test shows that the test was positive in eleven out of twelve clinically positive cases, and in four cases in which the evidence was inconclusive. It was negative in three cases in which other evidence of cure was insufficient, but in seven others the clinical findings were corroborative. We are of the opinion that for the purpose of establishing a cure, the test is of considerable value.

A year later, the same author[7] strongly opposes Spaulding's views of the frequency of relapses, arguing that in her cases "the test of cure was insufficient." He writes that "if after three or more months, during which frequent smears from the depths of the vagina have contained no pus, and all other signs of the infection have disappeared, a discharge containing gonococci recurs, we believe that this attack is frequently a reinfection, very possibly from the same source as the first."

R. M. Smith[2] says that "when the original source of infection still exists and no reform has taken place in the habits of the patient, a reinfection may easily occur and seems . . . more likely than that a relapse has taken place."

GONOCOCCUS INFECTION IN BOYS

Smith and McKee[16] report a case of urethritis in a boy of 17 months. Gonococci were found in smears and in culture. The source of infection was probably the nurse.

Nicoll and Wilson[17] describe a case of arthritis in a boy of 2½ years, following scarlet fever. Two of the joints became purulent and had to be opened. To their great surprise gonococci were found in the culture. Blood cultures were negative. The urethra appeared normal in every way, but two out of five cultures taken from it contained gonococci.

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15. Smith, George G.: The Complement Fixation Test in the Management of Gonococcus Vulvovaginitis, Am. Jour. Dis. Child., April, 1913, p. 313.
16. Smith, A., and McKee, C. S.: Brit. Med. Jour., 1913, i, 878.
17. Nicoll, M., Jr., and Wilson, M. A.: Jour. Infect. Dis., 1913, xii, 52.

PYELITIS

Mode of Infection

Thomson[18] in an excellent paper based on seventy-one cases of colon infection of the urinary tract in his private practice, says:

It is scarcely ever possible to decide in any individual case which route the bacilli takes in passing from their original harmless position to where they ultimately set up disease. It is, however, quite certain that they are sometimes carried there by the blood stream, sometimes pass in by the lymphatic channels, and sometimes ascend from outside by the lumen of the urinary tract.

When the bacilli reach the urinary tract . . . they are either washed out immediately . . ., or else destroyed . . .; or they may remain for a time doing little or no harm. . . . If the virulence of the organisms has been increased by morbid processes in the intestine, or otherwise, or the normal resistance of the tissues lowered by local or general disease; or perhaps if there has been some retardation of the flow of urine . . ., then more or less violent inflammation . . . may be set up.

He then analyzes the sex incidence of the disease, making the following points:

1. . . . the number of girls is much larger than that of boys; among my cases the girls formed 79 per cent.

2. In the young infants . . . the disease begins more often with diarrhea than in older children. . . . Diarrhea is more frequently a marked symptom in boys than girls. [That is] cases setting in during apparent good health are commoner in girls than in boys.

3. Further . . . during the first six months of life far more boys than girls are affected, although after the sixth month girls largely predominated. When we . . . analyze the ages and sexes of 224 babies under 2 years taken at random from thirteen authors we find that . . . there is still a considerably greater proportion of boys during the first six months than at any later age.

4. The extreme rarity of rigors at the onset . . . in boys is noteworthy . . . when compared with the frequency of this symptom in girls . . .

5. Mild cases of *Bacillus coli* infection are rare in boys in my experience. In male patients the attacks of pyelitis are apt to be severe, and there is usually in them a much larger proportion of cases of fatal pyelonephritis than among girls.

In discussing these five points, he writes:

. . . the great preponderance of female patients seems . . . to force us to the conclusion that ascending infection by way of the urethra must be a common occurrence; and the excess of girls over boys probably represents the frequency with which urethral infection takes place. It seems very unlikely that the colon bacilli can force their way up the lumen of the male urethra. The greater prevalence of antecedent diarrhea in boys would probably cease to exist if we were able to subtract from the list of girls the primary cases infected *per urethram.*

The frequency of pyelonephritis in the male sex may be explained by the infection having usually in them passed straight from the bowel to the kidney and pelvis, and not having ascended from below. The facts that rigors are so very rare in acute pyelitis in boys, and in pyelonephritis in both sexes, while they are so common in acute pyelitis in girls (in whom we suspect an ascending infection) suggest the idea that the ureters may be the particular portion of

18. Thomson, J.: Lancet, London, 1913, ii, 467.

the urinary tract from irritation of which a rigor most readily arises. The frequency with which rigors are met with in ureteral calculus may be held to support this view.

Somewhat similar views are held by Wyman,[19] who, from a study of sixty-five cases at the Children's Hospital in Boston, concludes that the ascending route is the common one in girls, and the hematogenous in boys, while what he calls the transparietal is rare in both sexes. An analysis of the age and sex of his patients gives the following:

Males under 2 years	= 37 per cent.
Females under 2 years	= 63 per cent.
Males over 2 years	= 10 per cent.
Females over 2 years	= 90 per cent.

The views of F. H. Smith[20] are less clear; he writes that "in the younger children and babies the infection is apt to be the colon bacillus, and most often from direct fecal contamination of the vulva, or possibly, by more direct route through blood or lymph paths to the kidney, upset bowels preceding the illness being highly suggestive of increased toxicity of the intestinal flora." In older children, however, he considers that "repeated attacks of acute tonsillitis, acute bronchitis and 'kidney trouble,' in sequence, suggest their interdependence."

Freeman[21] believes in the ascending road of infection, because of the predominance of female cases.

Green,[22] however, writes that pyelitis "probably proceeds by lymphatic extension from the intestine," while "acute inflammatory nephritis . . . is most usually hematogenous in origin." Nevertheless he includes as predisposing causes "calculi, constipation, phimosis, anal fissures and foci of infection elsewhere."

Cannata and Caronia[23] emphasize the necessity of preventing the early infections. Habitual constipation is dangerous because there is usually elimination of bacteria by way of the urinary apparatus. Fecal contamination of the vulva, phimosis and foreign bodies in the bladder, are the sources of ascending infections. Chilling, or any trauma to the bladder, is likely to prove an exciting cause. Stiner[24] also mentions the dangers of chilling.

Langstein[25] has reported two cases of pyelitis complicating Hirschsprung's disease, and also as a sequel to pertussis a fatal pyelitis due to Friedlander's bacillus. He writes that "in the future the hematogenous

19. Wyman, E. T.: Boston Med. and Surg. Jour., 1914, clxx, 540.
20. Smith, F. H.: Old Dominion Jour. Med. and Surg., 1914, xix, 77.
21. Freeman, Rowland Godfrey: Urinary Analysis in the Diagnosis and Treatment of Diseases of Infancy and Childhood, Jour. Am. Med. Assn., Nov. 21, 1914, p. 1802.
22. Green, R. M.: Boston Med. and Surg. Jour., 1913, clxviii, 645.
23. Cannata, S., and Caronia, G.: Pediatria, 1914, xxii, No. 9.
24. Stiner, O.: Cor.-Bl. f. schweiz. Aerzte, 1914, xliv, Nos. 23-26.
25. Langstein, L.: Med. Klin., 1913, ix, 1491.

and lymphatic mode of infection will be placed more in the foreground."

Kowitz[26] is practically alone in believing that the ascending route of infection is uncommon as compared with the hematogenous or lymphatic. He was led to this opinion first by finding that his own cases did not show the customary predominance of girls; in forty cases he had seventeen males to twenty-three females. "This does not seem to be such an overwhelming predominance of females that we can speak of an essential difference between the sexes." He then discovered in his cases the marked relation to the time of year shown in the accompanying chart.

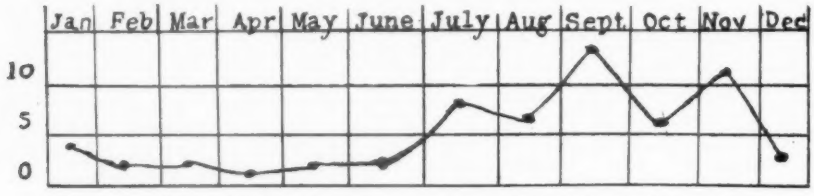

Chart showing relation of occurrence of pyelocystitis to time of year.

To prove pyelocystitis a descending hematogenous disease, it is necessary to find a source of infection elsewhere in the body; that is, the acute digestive upsets of the summer. Pyelitis does not accompany, but follows the diarrheas. The frequency of colon bacillus infection is to his mind another proof of the hematogenous mode of infection.

Clinical Symptoms

Wyman[19] has analyzed carefully the symptoms occurring in his sixty-five cases and gives the following list:

Vomiting	in 39 per cent.
Dysuria	in 36 per cent.
Pollakiuria	in 40 per cent.
Chills	in 15 per cent.
Abdominal pain	in 16 per cent.

Furthermore, he has found that pyelitis occurred as a complication of other disease as follows:

Otitis media	8 times	Pneumonia	7 times
Bronchitis	2 times	Infectious diarrhea	4 times
Typhoid	2 times	Intestinal indigestion	2 times
Measles	2 times	Tuberculous peritonitis	1 time

Gordon[27] separates the cases according to their clinical signs into these five groups.

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26. Kowitz, H. L.: München. med. Wchnschr., June 16, 1914, p. 1341.
27. Gordon, R. G.: Brit. Jour Child Dis., 1914, xi, 252.

1. Symptoms of general feverish disorders, without any indication that one special symptom was affected.
2. Cerebral symptoms.
3. Pulmonary symptoms.
4. Abdominal symptoms.
5. Urinary symptoms.

Langstein[25] has a similar classification based on the dominating clinical symptom; that is, a toxic type, a meningeal, a pulmonary, a gastro-intestinal type, one associated with icterus, and one which gives most of the signs of appendicitis. He also mentions a form of pyelitis in which the urine contains many red blood corpuscles and only a few pus cells and says that this "is frequently the cause of erroneous diagnosis."

Thomson[18] and Marsh[28] both emphasize the frequency of chills or rigors, the former saying that he does "not remember ever having seen an infant under 2 years who had had a well-marked rigor and who had no pus in the urine"; and the latter, that "shivering . . . is exceedingly rarely met with in infancy, except in this condition."

Stiner[24] mentions redness and swelling of the external genitals together with inability to urinate, as the initial symptoms of pyelitis which is brought on by exposure to cold.

Urinary Findings

Langstein's[25] hemorrhagic type must be again mentioned, the sediment consisting of "many red blood corpuscles, only scattered leukocytes, and perhaps here and there a cast. At the time it is difficult to decide whether this is the picture of a hemorrhagic nephritis, a calculus or a beginning pyelitis. . . . There are cases which proceed directly to recovery, provided proper medical treatment is instituted at once."

Freeman[21] endeavors to draw a line between a normal and a pathological number of pus cells in the urine. According to him "1-2 leukocytes in a D-field (Zeiss) of precipitate [of centrifugalized urine] from a female baby does not indicate a pyelitis . . . That number in a male baby, or more in a female baby, should render the diagnosis suspicious." F. H. Smith[20] has a somewhat similar standard, although he does not find a difference between the sexes. He writes that "it is by no means exceptional to find as few [pus cells] as 3 to the ⅙ field, and yet the symptoms be most severe."

Kowitz[26] has isolated the infecting organism in forty-four cases and confirms the predominance of the colon group, thus:

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28. Marsh, N. P.: Brit. Jour. Child. Dis., 1913, x, 385.

B. coli	24
B. paracoli	10
B. lactis aerogenes	7
Staphylococcus (cases of eczema and furunculosis)	3
Mixed	3

Other authors have nothing new to add to the already well-known urinary picture.

Duration of the Disease

Thomson,[18] Freeman,[21, 29] Marsh,[28] Wilson[30] and others, agree that the disease may last untreated for several years and show few or no symptoms. It may flare up at any time, however. With correct treatment, Langstein[25] considers that 90 per cent. of the patients recover. In severe cases the patients may recover completely after three and one-half years, or the condition may lead to a contracted kidney or to a nephrosis. Marsh[28] gives a favorable prognosis, but adds that "before pronouncing the patient cured, the urine should not only show an absence of organisms and of pus cells, but should also be culturally free . . ." "In spite of all treatment, many cases become chronic; in these acute exacerbations alternate with long periods of quiescence, but the urine never becomes entirely free from pus cells and organisms."

Treatment

The one form of treatment against which no dissenting voice is raised is the administration of large quantities of fluid. Further than this, there is apparently no specific treatment. In many of the acute cases the patients get well untreated.

Thomson[18] gives sufficient potassium citrate to maintain an alkaline reaction in the urine. Marsh[28] and Wyman[19] agree with this procedure. The latter, however, favors changing the reaction of the urine back and forth by the alternate administration of potassium citrate and sodium benzoate. Freeman[29] says that "the alkaline treatment of pyelitis, while it is safe and will control many cases, is markedly less efficient than other methods of treatment. Langstein[25] goes so far as to say that alkalies are useless in the treatment of the disease.

Probably the best of the so-called "urinary antiseptics" is hexamethylenamin. It is pretty generally agreed now that it acts only in an acid medium. Freeman[21, 29] considers it an "absolute antiseptic for certain infections of the urinary tract." He begins always with small doses of from ½ grain to 2 grains several times a day, and then increases rapidly, being careful to watch the child and its urine for symptoms of irritation of the kidneys. "Large doses of hexamethyl-

29. Freeman, Rowland G.: The Diagnosis and Treatment of Pyelitis in Infancy, Am. Jour. Dis. Child., August, 1913, p. 117.
30. Wilson, H. W.: Brit. Jour. Child. Dis., 1913, x, 289.

enamin should not usually be continued for more than a week at a time, and then after several days without any treatment, or with alkaline treatment, it should be started at the maximum dose given before and the amount increased daily until an influence on the urine is obtained. Doses of 25 grains daily in a child of 6 months, and 35 to 45 grains a day in a child of 9 to 12 months, may be safely given in this way to some infants." Wyman,[19] as second choice to the alkaline treatment, gives hexamethylenamin and sodium benzoate. Cannata[23] uses hexamethylenamin alone or alternating with phenyl salicylate (salol), offering at the same time large amounts of an acid drink such as diluted hydrochloric or phosphoric acid. Marsh[28] and Thomson,[18] representing the English point of view, are sceptical about the value of hexamethylenamin and phenyl salicylate. Kowitz[26] considers all urinary antiseptics useless, while Langstein[25] admits that hexamethylenamin may be of value as a prophylactic, but that "in severe cases more is accomplished by salol, in not too small doses (0.6 gm. to 1.0 gm. per diem)."

The value of vaccine therapy is still much disputed. Freeman,[29] writing in 1913 of renal infections with the colon bacillus, says: "Vaccines, either autogenous or commercial, are useful in controlling the constitutional symptoms of pyelitis." After another year's experience, he adds[21] that they "produce a reduction in the temperature; but, so far as I know, the vaccines do not cure the disease, although they apparently cure it. The child loses its fever and will increase in weight and seem perfectly well, but when the urine is examined one finds the same pus and bacteria as before." Green[22] and Wyman[19] also advise vaccines, but only in resistant cases. Marsh[28] and Kowitz[26] are doubtful about their value. Wilson[30] reports a case in which autogenous vaccines were used without success. The parenchyma of the right kidney finally became involved, abscesses formed, and a nephrectomy was done which was followed by prompt recovery. Thomson[18] reports that he has "used vaccines in a few cases, but with disappointing results." Langstein[25] has not only never seen good results from vaccines, but has also "seen a child on the second injection suddenly become very sick, with a high temperature and bloody stools." He considers that this may have been very probably an anaphylactic phenomenon.

TUBERCULOSIS

Renal tuberculosis in children is a very rare disease. Oraison[31] reports three new cases, all in girls, bringing the total number of reported cases up to fifty-one. One of his patients had a severe Pott's

31. Oraison, J.: Jour. d' urol., 1913, iv, 15.

disease, was not operated on, and died; the other two had nephrectomies; one apparently recovered and the other was lost sight of. Rocher and Ferron[32] report a case, also in a girl, in which the offending kidney was removed with resultant cure. For diagnosis both articles advise cystoscopy and ureteral catheterization. It is surprising at what an early age this is possible, that is, in girls after 5 years,[32] and in both sexes after 6 or 7 years.[31] "The urethra admits easily a No. 40 tube 7 cm. long, and, in spite of the reduced field, the shortness of the tube permits the exploration of the bladder . . ." "The ureteral opening admits usually a No. 6 or No. 7 sound." "We have frequently employed general anesthesia, not because these maneuvres are painful, but because the children became very often unmanageable with fright at the sight of the instruments."[32] "Finally, the treatment of renal tuberculosis in childhood presents nothing unusual . . . It is better, even for an infant, to live with a single healthy kidney than with both kidneys, one of which is diseased and the other constantly threatened."[31]

Primary genital tuberculosis is also rare. Oliver Lyons[33] reports three cases in small boys. In two of these the infection was in epididymis and the patients appeared to be in perfect health. The onset was sudden with pain on palpation, edema of scrotum, effusion into the tunica vaginalis and enlargement of the testicle. "In the case in which the prostate and seminal vesicles were found infected, frequent urination was the first symptom noticed by the mother, but as the case progressed the irritability became so marked that the patient was unable to retain his urine more than twenty or thirty minutes at a time." Also, in contrast to the other two, he "presented a typical tuberculous appearance." From a study of his cases and of the literature, Lyons concludes:

> For the true conception of these cases it is important to know that tuberculosis of these organs remains almost always the only tuberculous focus in the body; differing from later periods of life when it is frequently secondary to other processes in the genito-urinary tract. Therefore operation may produce permanent cure, although this is not always certain.

Graefe[34] has analyzed the necropsy protocols of eighty cases in all ages which showed tuberculous lesions in the female genitalia. He found nineteen of these to be in girls under 15 years old, which is a surprisingly high number. He studied these nineteen cases further and found that in all the lesions in the genitalia were secondary to tuberculosis elsewhere in the body. The distribution of the lesions was as follows:

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32. Rocher, H. L., and Ferron, J.: Jour. d' urol., 1913, iii, 153.
33. Lyons, Oliver: Tuberculosis of the Genital Organs in Children, Jour. Am. Med. Assn., Dec. 6, 1913, p. 2051.
34. Graefe, G.: Monatschr. f. Geburtsh., 1914, xl, 448.

	No. Cases		Per Cent.
Uterus only	0	=	0
Cervix	0	=	0
Tubes only	5	=	26.3
One tube only	3	=	19.2
Both tubes only	2	=	10.5
Uterus and both tubes	14	=	73.7
Both ovaries	3	=	19.2
One ovary alone	0	=	0
Uterus, both ovaries and both tubes	3	=	19.2
Vagina	1	=	5.3
Vulva and clitoris	0	=	0

"This table shows that in girls as in women the tubes are the most frequent site of genital tuberculosis."

The same cases classified according to age, give this result:

Under 1 year	1 case
1- 5 years	9 cases
5-10 years	2 cases
10-15 years	7 cases

The author has no explanation of the high figure for the 1 to 5 year period. The increase after 10 years, however, is probably due to the influence of puberty.

Holt[35] has reported a case of miliary tuberculosis following a ritual circumcision:

> A healthy child, born of healthy parents, breast fed, developed local symptoms of infection within a few days after the operation and these persisted, being followed after a few weeks by symptoms of general infection which continued until death. The lesions found at the necropsy point strongly to a spreading of the infection through the lymphatic system beginning from the wound, and afterward to a general blood infection.

He abstracts the 40 previously reported cases, and says that of the 41 patients, 16 are known to have died, 7 are tuberculous, in 12 the final results are not known, and only 6 are stated to have recovered. For the treatment, he advises the early removal of the inguinal glands. He concludes "that syphilis is less frequently acquired in this manner than is tuberculosis," and that "it is the duty of the physician to raise his protest against the performance of ritualistic circumcision in every case."

Champtaloup[36] reports from New Zealand a similar case, except that the operator was a physician who has since died of laryngeal tuberculosis. The child was 7 weeks old at the time of the operation. The wound suppurated badly and after three weeks an abscess formed in the right groin. Another small abscess appeared in the skin and the child became emaciated and hectic. Champtaloup first saw the child when it was 10 months old, at which time smears and cultures of the pus were negative, although guinea-pigs inoculated at the same time

35. Holt, L. Emmett: Tuberculosis Acquired Through Ritual Circumcision, Jour. Am. Med. Assn., July 12, 1913, p. 99.
36. Champtaloup, S. T.: Brit. Med. Jour., 1914, i, 814.

showed a well marked tuberculosis in four weeks. The patient was treated with tuberculin and when seen eight months later was the picture of health.

PSEUDOTUBERCULOSIS

Scherber[37] has described an acute inflammation of the vulva which leads to an ulceration resembling tuberculosis. It heals readily under cleanliness and mild antiseptics.

RHEUMATIC ORCHITIS

Bass[38] describes a boy 2½ years old who developed erythema nodosum and torticollis and four days later, while under salicylates, the scrotum suddenly swelled. "Examination showed the scrotum . . . twice its normal size. Marked local heat. Skin of scrotum edematous and dusky in color." The left testicle was enlarged and exquisitely tender, the right normal. The condition quickly disappeared and a month later there was "no atrophy of the testis."

329 Beacon Street.

37. Scherber, G.: Wien. klin. Wchnschr., 1913, xxvi, 1070.

38. Bass, M. F.: Acute Rheumatic Orchitis . . . Associated with Erythema Nodosum and Acute Torticollis in a Child Aged Two and One-Half Years, Jour. Am. Med. Assn., May 21, 1913, p. 1608.

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